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THE MAIN VARIETIES OF ARTERIAL DISEASES.[1]

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As your chairman has told you, I am on a holiday. When I left my native country I thought I was never to speak on a pathological subject again. However, I have been asked to give two addresses here and have agreed to do so with mixed feelings, as you can perhaps understand, because I have become extremely lazy and want to do nothing. At the same time I am pleased indeed to do anything that will meet the wishes of your Association. I want to say how pleased I am at the great amount of interest shown in Sydney in post-graduate instruction, in fact at the interest shown in it in the different parts of Australia I have visited. I think it a very good sign that those who have graduated should maintain their interest in the progress of the particular subjects to which they have applied themselves and should still be eager to hear more about them.

The fact that I did not anticipate having to speak on a pathological subject has prevented me from equipping myself as I might have done. I have no slides with me to illustrate the subject on which I am going to speak tonight, and that is a great disadvantage to myself as well as to you, because it is so very much easier to talk about things one can see.

[1] A lecture delivered in Sydney under the auspices of the New South Wales Post-Graduate Committee in Medicine on December 8, 1936.

The subject of my address is very wide, and, as you know, one which has many different aspects, each aspect involving a great deal of detail. My wish tonight is to express simply the main thoughts in my mind concerning it.

As a preliminary I should like to refer to one or two points with regard to the arterial system which must be kept in mind in studying this subject. As [illegible] known, arteries change through the different periods of life; there is the old saying that "a man is as old as his arteries". In the first place, the arteries of the young contain a larger proportion of muscle than do the arteries of those late on in life, or, to put it another way, the arteries of the aged are less muscular or more fibrous. Perhaps the term "less muscular" would be the more appropriate. This tendency towards a relative increase of connective tissue in the arteries with advancing years is an important point in relation to pathological changes.

The second point is that there is also a tendency to an increase of calcium in the artery walls in later years. This was worked out some years ago by Andrewes, who showed that in the aortic tissue, apart from the lesions of disease, the calcium content increased with age. Accordingly, in later life the aorta contains a larger amount of calcium which is ready at hand to be deposited. One is justified in supposing that the same holds with regard to other arteries.

A third point to be noted is that there is a very great tendency to degeneration associated with the deposit of lipoids in the intima of arteries, and this is the case throughout life. For example, in the aorta of the child it is quite common to find yellow streaks and patches in a variety of pathological conditions. If these are examined it is found that underneath the endothelium of the vessels there is an accumulation of lipoids, chiefly in the form of cholesterol esters, in the cells of the intima. Thus it is quite clear that that accumulation of lipoids, an indication of degeneration, occurs on what one might call slight provocation; it is met with, even in early life, in wasting diseases, acute febrile conditions *et cetera*. As we shall see later, this lipoid infiltration becomes greatly intensified in various conditions later in life.

The fourth point is that the arteries have a wonderful power of responding to the particular call made upon them. For instance, as you know, when an artery is tied the collaterals dilate, and in the course of time become hypertrophied; thus we have larger arteries. So, too, when there is long-continued high blood pressure, the arteries adapt themselves to that abnormal condition, and compensatory hypertrophic changes occur in their walls in a way to which I shall presently refer in greater detail. I have referred to these points because the changes mentioned—fibrosis, deposition of lime salts and lipoids, and compensatory changes—are the essential ones in arterial disease and are found in all combinations.

I may set out the main arterial diseases in the following way:

I. Non-Infective—
- Atheroma.
- Arteriosclerosis.
- Medial degeneration (calcification of media and medio-necrosis).

II. Infective—
- Syphilis.
- Rheumatism.
- *Polyarteritis nodosa.*
- *Thromboangiitis obliterans.*

Of these, atheroma, arteriosclerosis and syphilis are the most important and will form the subject of my lecture. I shall speak of the characters and main effects of each, and it will be more convenient if I consider arteriosclerosis before atheroma.

Arteriosclerosis.

There has been a great deal of variation in the use of the term "arteriosclerosis" and a great deal of confusion has resulted. I think this is largely due to the account given by German pathologists, who include under arteriosclerosis or, as some prefer to call it, atherosclerosis, not only diffuse fibrosis of arteries, but also the patchy degenerative lesion of atheroma. They fail, too, as a rule to emphasize the important relationship between general arteriosclerosis and hypertension. And I think the American pathologists have largely followed the German pathologists in this matter. This is mainly because of an attempt to study diseases from the purely structural point of view without regard being had to ætiology and the clinical conditions with which the structural changes are associated. British pathologists, on the other hand, have as a rule recognized for a considerable time the important difference between arteriosclerosis and atheroma; and I am pleased to find, what I did not previously know, that Professor Welsh here has for many years insisted in his lectures on the essential distinction between them. The difference is one which concerns not only the nature and evolution of the structural changes, but also the conditions of occurrence and especially the results which follow from the lesions.

I might put the matter briefly at the present stage by saying that arteriosclerosis is a diffuse lesion of arteries attended by stiffening or hardening, and this is essentially due to fibrosis, an increase of the connective tissue in the walls of the arteries, which, in the great majority of cases, follows on hypertrophic changes due to high blood pressure. On the other hand, atheroma is a localized or patchy lesion; essentially a degenerative lesion, it is primarily an affection of the intima, whereas in arteriosclerosis all the coats of the arteries are affected. These few points of difference serve sufficiently for the present to distinguish the two types of disease of which I am speaking. We have now to consider arteriosclerosis in more detail.

How are we to study it? How are we to study any particular form of arterial disease? Our endeavour should be to get, as far as possible, examples of the disease in its purest form. If one

takes the case of a man pretty well up in years who has had arteriosclerosis with increased blood pressure, he may likely have also atheroma, and in addition he may have also been the subject of syphilis. In a case like this it would be quite impossible to disentangle the various kinds of lesions present. We must then, as far as possible, get a pure example of the particular type of disease we wish to consider. For instance, in syphilis it is advisable to get an example of the disease occurring early in life, in a young man between twenty-five and thirty years of age, in whom there is very little chance of some other important type of arterial disease being present. We then get the syphilitic disease in its pure form, and we can work out its essential features. I shall speak of this later. We have thus to find the best sort of case in which to study arteriosclerosis, and we follow the general rule that the younger the patient, the more likely are we to get the disease in its pure form.

The commonest form of arteriosclerosis is that associated with high blood pressure. We may speak of it as hypertensive arteriosclerosis, and this is by far the most important form of the disease. It is true that in very old people one finds a general fibrosis of arteries or arteriosclerosis without any rise in blood pressure. But these are patients approaching the natural termination of life, and in them the lesion is not of the same importance.

How, then, are we to study best the condition of thickening of the arteries associated with high blood pressure? As I previously remarked, the younger the patient, the better. The best example that we can get, in my opinion, is a patient who has had an initial lesion of the kidneys, an acute nephritis at the age of twenty or earlier, an affection of all the renal elements, the glomeruli, tubular epithelium and interstitial tissue, accompanied by elevation of the blood pressure. The patient appears to recover and the high blood pressure disappears for a time; but later it begins to rise again and the hypertension persists. Such a patient may die from uræmia or some other complication, and one can then study the condition of the vascular system, which has thus resulted from the prolonged hypertension.

In a case like this I think we are quite justified in assuming that the changes we find in the arterial system are the result of the hypertension, especially as they receive a rational explanation on this basis. There are, of course, in nephritis various biochemical disturbances, but there is no evidence that they have any action on the arteries except through the vasomotor mechanism. Initially, of course, the high blood pressure is the result of constriction of the arterioles. But as the condition proceeds, this constriction is followed by hypertrophic changes in the walls of the arteries—that is to say, changes which are adapted to meet the increased tension placed on the arteries by the high blood pressure.

Some years ago I made an investigation of the arterial changes at different stages when this high blood pressure had been produced by acute and subacute kidney diseases. The results were very definite. After the constriction has lasted some time there occur compensatory hypertrophic changes which concern both the muscular and the elastic elements. The muscle fibres in the media show an increase in number, the coat thus becoming thickened; and it is interesting to find that the few longitudinal muscular fibres situated in the intima also show hypertrophy, sometimes of striking degree. These longitudinal fibres in the intima resist elongation of the vessel, which is apt to occur when there is increased arterial pressure. This increase both of the circular fibres in the media and of the longitudinal fibres in the intima of course represents an adaptation to meet the tendency to dilatation and elongation respectively of the vessel due to the increased pressure within it. At the same time there is an increase in the elastic tissue of such a vessel. There is a splitting off of new elastic laminæ from the internal elastic lamina, and there is an increase of elastic tissue also in other parts of the vessel. These changes are hypertrophic, but after a time they begin to fail; both muscle fibres and elastic tissue begin to show degenerative changes. Nature then does the best it can by replacing the degenerating tissue with fibrous tissue, and as time goes on the hypertrophied artery wall becomes more and more fibrous. Accordingly, when we study the development of arteriosclerosis in primary kidney disease we make out these three distinct stages: first of all, contraction of the arterioles (functional vaso-constriction); secondly, hypertrophic change in the artery walls; and, thirdly, failure on the part of the hypertrophic elements with progressive increase of fibrous tissue, the ultimate result being a fibrosed artery.

Arteriosclerosis may thus result from a lesion primarily renal; therefore we can speak of a "secondary arteriosclerosis", that is, one secondary to renal disease. But, as you know, that is not the usual mode of occurrence of arteriosclerosis; in most cases it develops in an insidious way without any preceding kidney lesion. This is the state of arterial fibrosis which is found in the clinical condition now called essential hypertension, though we do not know what "essential" signifies. This is a good term. Unfortunately we have not a correspondingly simple term for the accompanying structural changes. We may speak of the condition as primary arteriosclerosis, in contrast with the form secondary to renal disease, but "primary", of course, is not strictly correct. The term, however, may be used as a convenient one. To be quite accurate one might call it "primary hypertensive arteriosclerosis" or "primary post-hypertrophic arteriosclerosis", but these terms are inconveniently long. Some writers use the term "arteriolosclerosis", but this overlooks the fact that all the arteries are affected. We know that this condition is extremely common, that it develops in an insidious way. The changes in the arterial tree are of the same nature as those which are found after an initial renal lesion, but the

fibrotic changes as a rule predominate. When one comes to examine a case, one finds that the hypertrophic changes have largely passed away. There is fibrosis of the media and there is also general thickening of the intima, which varies greatly in different arteries.

Now I may add a word or two with regard to the state of the arteries in this condition of primary hypertensive arteriosclerosis. The whole arterial tree is affected. The large arteries are larger than normal and their walls are thicker. Especially is this noticeable in the visceral arteries and other branches of the aorta. If you dissect the aorta and its branches, the latter stand out prominently, needing no support, whereas normal vessels collapse. Furthermore, the larger and medium-sized arteries have an increased lumen as well as increased thickness of wall. The aorta also is hypertrophied; it is a heavier aorta than that of a normal subject. Whilst these are the changes in the larger arteries, we find that a number of the smaller arteries are the seat of intimal changes which are partly of the nature of hyaline change and partly of the nature of an obliterative process. They result essentially in the diminution of the lumen and are commonest in viscera, especially the kidneys. I shall presently refer to them more in detail. Along with these changes in the arterial tree there is, of course, the great hypertrophy of the left ventricle due to the increased peripheral resistance. *Post mortem* examination accordingly supplies us with a picture of a heart of greatly increased power, related to an arterial system which has developed greatly increased powers of resistance to hypertension. That is the picture which the clinician should have in his mind when a case of hypertensive arteriosclerosis comes before him.

I think we are justified in believing that the sequel of events is the same in this primary arteriosclerosis as it is in the secondary form, that is, due to primary kidney disease. This means that here also we have the three stages: firstly, the contraction of the arterioles; secondly, hypertrophic changes in the arteries generally; and thirdly, an increase in fibrous tissue leading to sclerotic stiffening of the artery walls. As I have said, in most instances the fibrotic or post-hypertrophic changes preponderate; medial fibrosis is a prominent feature and the intima shows degrees of thickening even in the large arteries. There are also the changes in the arterioles, already mentioned, and these vary greatly in their distribution. They are practically always present in the kidneys and are associated with a diminution in the lumen of the affected vessels; they are common also in the spleen and in other parts of the body. (Of course it must be admitted that our knowledge regarding the arteries in the various parts of the body is very slight indeed, because a *post mortem* examination, however thorough, hardly includes an examination of all the arteries in the body.) It is difficult to say what produces the changes in the small arterioles. I think it quite likely that the vaso-constriction of the arterioles in certain areas interferes with their nourishment, and in this way the hyaline alteration and intimal thickening may be brought about; but the relation of these changes to the high blood pressure is certainly a problem, as is also their variation in distribution.

One now passes on naturally to consider shortly the condition of the kidneys in primary arteriosclerosis, the renal changes being of secondary nature. The lesions vary greatly according to the extent to which the renal arterioles are affected. Sometimes they are affected very little, the kidneys being of large size and having a smooth surface, though often they are the seat of chronic venous congestion. Sir William Osler was one of the first to point out that in essential hypertension the kidneys might be little altered in size and naked-eye appearance. But in all cases, even when the kidneys are large and appear smooth on the surface, you will find changes in the small arterioles. These changes lead to secondary nutritional changes in the glomeruli, and in this way the latter are progressively thrown out of functional activity. In other cases the kidneys are partly shrunken and show irregularities, while in others again they are in the typical "granular contracted" state, though this condition is, in my experience, not so common as is generally supposed. These variations in the general state of the kidneys depend on the degree to which the arterioles and, secondarily, the glomeruli are affected, but one cannot explain why this factor should vary so much. The condition of the kidneys acquires importance in proportion to the interference with renal efficiency, to which further reference will be made.

I have mentioned the hypertension and the corresponding changes in the arterial tree and also in the heart. Many cases of essential hypertension run a very chronic course, and the patient, as you know, dies either from cerebral hæmorrhage or as the result of cardiac failure. I do not need to say anything about cerebral hæmorrhage, as you are quite familiar with it. An important factor in the clinical course of the disease is the state of the left ventricle. As time goes on this hypertrophy may fail and be succeeded by dilatation, incompetence of the mitral valve, general chronic venous congestion, œdema *et cetera*; that is to say, a patient may end with the ordinary signs and symptoms of cardiac decompensation. The cardiac state is often influenced by the condition of the coronary arterial branches, fibrosis or actual infarction resulting according to the degree of closure. In this connexion it is to be noted that there is a tendency for patchy atheroma to supervene in hypertensive arteriosclerosis. It is important to bear in mind that, although the kidneys become damaged in varying degree, in many cases there is no actual interference with their functional activity. One often finds cases in which the structural changes in the kidneys are very pronounced, but in which there is no evidence of renal hypofunction, no increase of urea and other nitrogenous substances

in the blood, and no tendency to uræmia. Cases of this type are nowadays often spoken of as examples of simple or benign hypertension, and death usually comes about in one of the two ways mentioned.

It has, however, come to be recognized that in a relatively small proportion of cases of essential hypertension the disease assumes a more serious and more rapidly fatal character. This is evidenced by changes in the urine and in the excretory activity of the kidneys. The blood urea percentage rises gradually and there are other evidences of interferences with renal function; albumin, often with blood, appears in the urine and after a time uræmic symptoms develop; ultimately the patient dies of uræmia. That is to say, renal symptoms become superadded to those which are ordinarily associated with hypertension. In recent years the term "malignant nephrosclerosis" or "malignant hypertension" has been introduced to indicate this type of the disease, in contrast with the commoner benign type. I have examined kidneys from a considerable number of patients with this malignant type of hypertension and have found that the changes in the arterioles are more pronounced than in the benign cases. There is evidence of disturbance of the circulation, especially through the glomeruli. This is shown by the extremely irregular congestion which is present, and also by the presence of hæmorrhages in various parts of the kidneys. Necrosis of the walls of the arterioles is not infrequently found. Why should these renal changes follow in certain cases? I think we have rather overlooked the functional aspect in this type of disease. It seems to me that many of these changes in the kidneys may be the result simply of unusually great contraction of the arterioles. Whatever the agent concerned is, it undoubtedly affects the arterioles, and this affection varies greatly. If the contraction be excessive, one can understand how it is that secondary changes occur in the intima of the small vessels, those hyaline and obliterative changes which are so striking a feature. These changes become especially marked in the malignant type; the circulation is more interfered with, and the function of the kidney ultimately becomes seriously hampered. Much more investigation will be necessary before any definite conclusion is arrived at on this subject.

To sum up, then, this condition of primary arteriosclerosis, the lesion of essential hypertension, may bring about serious results in the first place, in a more or less mechanical way, by leading to cardiac hypertrophy followed by dilatation or by leading to cerebral hæmorrhage, or in the second place by bringing about serious impairment of the kidney function, resulting in the condition known as malignant nephrosclerosis; the case then becomes one of renal type.

With regard to the causation of primary arteriosclerosis, we can speak only in a general way. The essential and primary abnormality, as I have said, seems to be excessive vaso-constriction of the small arterioles in different parts of the body. While no one definite cause of this is known, certain factors appear to be concerned. One thinks in the first place of abnormal metabolism. For example, we know that hypertensive arteriosclerosis is apt to develop in cases of gout and lead poisoning. Such cases, however, form but a very small proportion of the whole, and it would at present be mere speculation to refer the general vaso-constriction to metabolic disturbance. Any condition leading to a dilatation of the arterial system will tend to increase arteriosclerosis once it has become established. Thus in cases of excessive eating and drinking the vessels become greatly dilated from time to time, thus imposing special strain on their walls. In fact, one sometimes finds cases of arteriosclerosis in which the changes are more pronounced in the mesenteric arteries and their branches than in any other parts of the body; that is rather suggestive. But we cannot rank such a factor as a general exciting cause. We know, too, that physical strain is an intensifying factor. In Glasgow, for instance, arteriosclerosis is very common indeed amongst men who are engaged in manual labour involving strain. But on the other hand, what are we to make of the well-known fact that this state of essential hypertension also appears in patients who are living quiet lives without any special strain of any kind? There is undoubtedly a tendency for hypertensive arteriosclerosis to run in families, and it is apparently hereditarily transmitted. But this factor applies only in a very small proportion of cases of the disease.

Abnormalities of the nervous system and the endocrine glands have also been brought into this question. We know that basophile tumours of the pituitary body lead to a symptom complex which includes hypertension—the syndrome of basophilism; and also that a chromaffine tumour (phæochromocytoma) of the adrenal medulla leads to hypertension, which, however, is of intermittent character. But all these facts seem quite insufficient in relation to the general question, seeing that this condition of essential hypertension is so common, whilst these variations in the endocrine glands *et cetera*, as far as we know, are not very frequently met with. At present, accordingly, we cannot say anything definite with regard to the real ætiology of the primary vaso-constriction which leads to arteriosclerosis. I might almost say that this is the burning question of clinical medicine. But I am not without hope that advances will be made in the future towards ascertaining what is the condition which leads primarily to the abnormal constriction of the arterioles.

I have spoken of hypertensive arteriosclerosis, that is, the general fibrosis of vessels accompanied by hypertension. There is another form, that met with in very old people, in which the muscle and elastic tissue degenerate and largely disappear. But this may be regarded as an exaggeration of senile involution and is really not of very much importance clinically.

Atheroma.

From the pathological point of view atheroma is a clearly defined condition. In contrast with arteriosclerosis, it is a degenerative change which starts in the intima and which is of a patchy nature, often leading later to secondary effects in the media. Especially is this the case in the smaller vessels, in which great atrophy of the media may be produced. In the intima there is an accumulation of lipoids which are chiefly of the nature of cholesterol esters, though some of the ordinary neutral fats are present too. Later the presence of cholesterol crystals and calcium salts is quite common. Along with the deposition of fatty material there is a varying amount of fibrosis, which occurs especially superficially to the degenerative material. There has been much discussion as to which comes first, the fibrous thickening of the intima or the deposition of lipoids. I have paid a good deal of attention to this point, and I find that I cannot draw any definite line between the fatty changes which occur in children and the more marked atheromatous changes that occur in adults. In children dying at quite an early period of life it is quite common to find yellow streaks and patches in the aorta, and if these are examined microscopically they will be found to be due to the accumulation of lipoids in the cells within the intima, not in the surface endothelium as it is sometimes described. Later in life we find the same change commoner and more pronounced, and later still the accumulation of lipoids is attended by a varying amount of fibrous thickening of the intima which is to be regarded as of a secondary nature. One frequently finds appearances which are incompatible with the opposite view, namely, that a fibrous patch is the first lesion and that then the lipoids are deposited in its deeper part as the result of impaired nutrition. It is not necessary to go further into details of histology; it is sufficient to give this general expression of my views.

The effects of the lesions, too, are essentially different from those of arteriosclerosis, as I shall presently describe. Atheroma is so common that one might almost call it normal after a certain period of life. It is the rarest thing to find a complete absence of atheroma in anyone in the middle or later periods of life. It is of importance only when it becomes of marked degree and leads to stenosis, thrombosis *et cetera*. There is very often an associated degeneration in the media, sometimes calcification of the media. Atheroma leads to a different collection of effects from those produced by arteriosclerosis. It varies greatly in its distribution, and it is curious how one sometimes finds very extreme atheroma in the medium-sized vessels and relatively little in the aorta, whereas sometimes the converse condition is found. Atheroma is extremely common in the aorta, especially in its lower part. It is common also, sometimes in severe degree, in the iliac and in the femoral arteries, and also in the arterial branches of the lower limbs. Of the visceral arteries, the coronary arteries of the heart and the cerebral arteries are probably the most frequent sites, and in both of these atheroma is often responsible for serious results. The occurrence of atheroma in the pulmonary arteries in obstructive conditions of the pulmonary circulation is a point of importance and will afterwards be referred to.

Let us now examine the main effects of atheroma from the clinical point of view. In the first place I may say there is no evidence, as far as my experience goes, that atheroma leads to hypertension and hypertrophy of the heart; one finds cases of extreme degree with a heart of normal weight. Another point is whether high blood pressure leads to atheroma; and this I shall discuss presently. What results are due to atheroma of the aorta? When severe and affecting the arch, atheroma often produces dilatation of the vessel. I believe it very rarely leads to aneurysm, though this may happen. Occasionally it spreads to and implicates the aortic valve, leading to incompetence, and at times the orifices of the coronary arteries are involved in an atheromatous patch and narrowed; in this way the nutrition of the cardiac muscle is impaired and diffuse fibrosis may follow. These two last effects, however, in my experience are not so common here as in syphilitic diseases. Thrombosis sometimes may occur on an atheromatous patch in the aorta and be the source of embolism. Of atheroma of the visceral arteries undoubtedly the most important effects are those seen when the cerebral vessels or the coronary arteries of the heart are affected. As far as the cerebral vessels are concerned, the commonest serious result is thrombosis, but rupture occasionally occurs. Cerebral hæmorrhage may be from an aneurysm developing on an atheromatous branch at the base of the brain, or may be in the substance of the brain. In the latter situation it is met with in very old people and occurs apart from any hypertension. The effects of atheroma of the cerebral vessels are undoubtedly serious, and equally serious, and even more common, are those produced by atheroma of the coronary arteries of the heart.

A great deal has been written in recent years about coronary thrombosis. It is extraordinary how, when one man starts to write on a subject, others follow; and the literature on coronary thrombosis has now reached very large proportions. An atheromatous vessel may become closed by thrombosis and the effects will be in proportion to the size of the vessel. I think they may be arranged in this way. When a large branch is closed, sometimes sudden death occurs, sometimes very grave symptoms supervene and death is delayed for a few hours or longer. In the latter event infarction of the cardiac muscle is found in a stage corresponding to the duration of the closure. Sometimes again there is a gradual impairment of the cardiac action with exacerbations, and when the patient ultimately dies one finds the infarctions in varying stages of absorption. Again, in other cases the narrowing of the small arteries leads to what is called a starva-

tion atrophy of the cardiac muscle, and this is followed by an overgrowth of connective tissue, so that patches of fibrosis are formed in the myocardium. This condition, in my opinion, is even commoner than actual infarction due to thrombosis. It may lead to gradual heart failure or occasionally to sudden death.

There is, however, one fact that is not sufficiently recognized, namely, that the narrowing of a coronary artery due to atheroma may cause sudden death without visible lesion of the cardiac muscle. I have seen many examples of this in Glasgow. The vessel most frequently concerned is the descending branch of the anterior coronary artery. This branch runs forward from its origin and on reaching the posterior aspect of the sternum curves downwards on the anterior wall of the heart. When there is a tendency to atheroma, you will often find that there is a marked patch with narrowing of the lumen just at the downward bend. This lesion apparently leads to impaired blood supply to the left ventricle, and when there is some sudden strain the blood supply becomes insufficient and fatal syncope occurs. Thus sudden death in atheromatous disease may be due to infarction, to fibrosis, or to such a vascular lesion without structural change in the myocardium. The effects of atheroma on the heart are thus various and of a very serious kind. Furthermore, they are of common occurrence.

I have mentioned that I think hypertensive arteriosclerosis tends to produce atheroma of the coronary arteries of the heart. It is not surprising that it should, because as the heart enlarges the coronary arteries have to increase in size too, and as they do so they probably become more susceptible to the conditions which bring about atheroma. The atheromatous disease of the coronary arteries then becomes a serious complication of arteriosclerosis.

The effects of atheroma on the kidneys, I think, are not of a serious kind. Branches of the renal arteries become implicated, the blood supply to certain areas of the kidney is interfered with, and irregular atrophy is a not uncommon result. But it is rare to find any severe functional effects produced by atheromatous disease of the renal vessels. It is scarcely necessary to add that atheroma may lead to thrombosis in any part of the body where it is of marked degree, for example, in the lower limbs, with resulting gangrene. That is very well illustrated in cases of diabetes, to be referred to later.

I shall now say a word or two regarding the ætiology of atheroma. There are two main factors concerned: one is the mechanical factor of strain, the other is metabolic disturbance. That a mechanical factor in the production of atheroma does come in is shown by its distribution in the aorta. It is often especially marked round the orifices of the intercostal and lumbar arteries, this being so much the case that it sometimes gives the appearance of a double row of buttons. That must be from a mechanical cause. Then there is the fact that atheroma as a rule is specially pronounced in the lower part of the aorta and that distribution is probably due to increased hydrostatic pressure in this region. Again, atheroma is common in the pulmonary arteries, for example, in cases of fibrosis of the lungs, emphysema, mitral stenosis *et cetera*. But that is not a clear case because there are other disturbances present, notably anoxæmia, imperfect oxygenation, which may play a part as important as the mechanical one. We have, on the other hand, the undoubted fact that arteriosclerosis may exist for a long period without leading to atheroma. I have studied this point in the earlier years and have no doubt that this holds good. I am pleased to find that Aschoff has come to the same conclusion. I think there is a good deal to be said for the view which he holds, that a sheering strain is of greater importance than simply increased pressure. You can understand how that at the orifices of the intercostal arteries there is a certain sheering strain, and this may tend to make the atheroma specially marked in that situation. Thus there is no evidence that increased pressure in itself produces atheroma, but there are facts which show that a purely mechanical factor does affect the distribution and intensity of the disease.

I may now refer briefly to metabolic factors in the production of atheroma. I have already stated that I cannot draw any distinct line between the fatty or rather lipoidal changes in the intima seen in children and the more marked atheromatous changes occurring in adults. Now the deposition of lipoids in the intima can readily be produced experimentally. If rabbits, for example, are fed with cholesterol dissolved in olive oil, the cholesterol is esterized in the bowel and absorbed and then becomes deposited as doubly refracting cholesterol fat in various parts of the body. A number of these experiments were carried out in our laboratory in Glasgow and the results were very definite. The cholesterol esters are deposited not only in viscera, such as the adrenals, liver, ovaries, testes *et cetera*, but also in the intima of the aorta. The deposition of lipoids in the last-mentioned situation may become very pronounced and may be followed by secondary proliferation of the connective tissue; thus a condition like atheroma may result. This opens up the possibility that abnormalities of metabolism in the human subject may quite readily lead to deposition of lipoids characteristic of atheroma. It is interesting, too, to note that in diabetes, in which there is a disturbance of the cholesterol metabolism, atheroma is sometimes very severe, especially in the arteries of the lower limbs, and, as you know, it not infrequently leads to thrombosis and gangrene. Other factors, such as the action of toxins, have been put forward, especially by French writers; but none of these factors can be said to stand in a definite causal relationship. It seems to me the position is simply this, that there is an inborn tendency for the deposition of lipoids in the arterial intima,

especially with advancing years, and that this tendency is increased by various conditions of strain and of abnormal metabolism, possibly also by the action of toxins and the like. There is, however, no one cause of atheroma. As already indicated, the commonest serious results are those arising from affection of the coronary arteries of the heart and the cerebral arteries; atheroma is thus responsible for a considerable mortality.

Syphilitic Disease.

There is no need for a lengthy discussion on syphilitic disease. In a sense it is the most definite form of arterial disease and the easiest to study. Here again for this purpose we select cases in which the arterial changes occur at a relatively early period of life. The arterial lesions of syphilis are readily explained on the basis that the spirochætes have a special tendency to gather in the adventitial connective tissue. Wherever they are present, they bring about the inflammatory reaction characteristic of syphilis; but the effect varies greatly according to the size of the artery affected. For example, in the aorta, where by far the commonest site of lesions is the arch, the cellular reaction, infiltration of lymphocytes and plasma cells, which starts around the *vasa vasorum*, extends into the media and leads to disruption and absorption of the elastic tissue and the muscle; occasionally there are patches of actual necrosis. There thus result weakened areas in the wall of the vessel. Where there is an area of weakening due to medial damage, the common occurrence is a reactive change in the intima leading to a laminated thickening. New vessels grow into the intima from the media, and I have never seen in an undoubted syphilitic lesion the deposition of lipoids characteristic of atheroma. Accordingly there occur in the intima of the aorta those raised greyish-white patches characteristic of syphilitic disease. The lesions in the media, of course, bring about weakness which often leads to local dilatation or to aneurysm. The same changes are met with in the larger branches of the aorta. But in the smaller vessels the irritation produced by the spirochætes in the outer coat is able to extend through to the intima and cause proliferative changes resulting in diminution of the lumen or actual obliteration of the vessel. The widely different results, dilatation of a large artery and narrowing or obliteration of a small artery, simply depend on the size of the vessel; the actual structural changes are of the same kind in both cases.

The chief effects of syphilitic disease of the aorta are threefold. In the first place there is the occurrence of aneurysm. I do not wish to speak of percentages, but there is no doubt that aneurysm of the aorta is, with few exceptions, the result of syphilitic disease. Aneurysm does occasionally follow as the result of atheroma, but on the whole it is rare. The second common result is implication of the orifices of the coronary arteries; and that leads to the usual starvation effects on the cardiac muscle—ventricular dilatation, diffuse fibrosis, intracardiac thrombosis *et cetera*. As has been said, atheroma of the aorta may lead to a like series of changes, though less commonly. The third effect is the extension of the chronic inflammatory changes to the aortic valve with serious effects on its efficiency. There may be a certain amount of adhesion of the cusps with stenosis, but incompetence is the all-important result. The valve segments become thickened, shortened or distorted; and another curious and serious change is that they sometimes seem to have lost their resisting power and become stretched and pendulous. Extreme aortic incompetence may be brought about in this way. Syphilis does affect the coronary branches, and may be associated with gumma or may occur apart from it. Some American writers find that syphilitic lesions in the myocardium are relatively frequent, but my own experience does not support this. (One must always bear in mind, however, that the incidence of pathological conditions varies in different localities.) They do occur from time to time, but I think that the important effects on the heart in syphilis are secondary to the lesions in the aorta, which act in two ways, namely, either by causing narrowing of the coronary orifices or by implicating the aortic valve.

As I said, smaller arteries tend to become obliterated. *Endarteritis obliterans* of the cerebral arteries is the most important example of this. Often attended by thrombosis, it leads to ischæmia and softening of the brain substance. But I need not discuss this. In text-books one often finds it said that cerebral hæmorrhage is produced by syphilis. I do not think this is correct. I do not say it is impossible that a syphilitic gummatous lesion should involve a cerebral artery and lead to rapid necrosis with subsequent hæmorrhage, but I have never seen such a case. The usual result is reactive obliteration, and I think for all practical purposes syphilis may be excluded as a cause of cerebral hæmorrhage. In this connexion I should like to add one important point, namely, that syphilitic disease of arteries does not cause any appreciable degree of elevation of blood pressure. In this respect it agrees with atheroma. To put the matter briefly, syphilis produces clearly marked arterial changes. It leads to a few definite lesions, the most important being those of the aorta on the one hand or the cerebral vessels on the other. The former are of common occurrence and are responsible for a considerable proportion of cardiac conditions of chronic type. It is important that the general features of syphilitic disease of arteries be distinguished from those of arteriosclerosis or of atheroma—three affections essentially different in their conditions of occurrence, distribution and effects, as well as in their histological changes.

Other Conditions.

These undoubtedly are the three main types of arterial disease—arteriosclerosis, atheroma and syphilis, and constitute the subject of my address.

In touching lightly, however, upon the others that have been placed in the table given, I may mention a few points. The senile condition of calcification of the media, often leading to thrombosis and gangrene, needs no description, but the lesion of medial necrosis has more recently been recognized. It is the extreme stage of a degeneration in the media and its importance is that it may give rise to dissecting aneurysm, the inner part of the media and the intima giving way, or to the rare event of spontaneous rupture of the aorta. In either event the intima may sometimes appear healthy. Nothing is known of the causation of the lesion in the media.

In the infective list I have placed rheumatism. The rheumatic lesions, although interesting from the histological point of view, are not of importance so far as clinical effects are concerned. It is sufficient to say that occasionally in rheumatism there are infiltrations of the outer coat of the aorta, somewhat of the same sort as those in syphilis, but they do not penetrate the media sufficiently far to produce aneurysm or any serious result. They have the same histological features as those in the heart and usually represent an extension of the latter. Rheumatic lesions in the smaller arteries have also been described, but I cannot speak of these from my own observations.

Polyarteritis nodosa was first recognized as a rare acute disease which leads to focal damage of the artery wall with formation of aneurysm and hæmorrhage; the lesions are multiple and especially affect the visceral arteries. In this form it has the characters of an acute infective disease. We have, however, come to recognize that it occurs more commonly as a subacute or chronic arteritis, which results in local dilatation, or more frequently in closure of the vessel, leading to infarction or fibrosis. These lesions are met with especially in the heart, kidneys and liver, and the condition is by no means rare. I have no need to say anything regarding *thromboangiitis obliterans*, as so much has been written about it in recent years. I have placed these two diseases, polyarteritis and *thromboangiitis obliterans*, provisionally in the infective group in view of their chief features, but whether this is justifiable or not can be determined only by subsequent investigation.

HYDATID DISEASE: A SURVEY OF SIXTY CASES.[1]

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IT is difficult to find any aspects of hydatid disease that have not already been dealt with exhaustively, and in many cases, it would seem, finally. Probably its incidence in Australia will gradually diminish; in fact, it would appear that this is already happening as a result of health propaganda and the establishment of central abattoirs in the larger country centres. However, in spite of propaganda and some control of slaughtering, the incidence of hydatid disease in sheep is still very high. Of 1,484 sheep livers examined during the last month at the Hamilton Municipal Abattoirs, 28·76% were infected with hydatid disease.

Most of the patients now affected in this district probably acquire the disease from contact with dogs that have become infested by feeding on the diseased offal of private and uncontrolled slaughtering. Moreover, it is just this source of infestation that public health propaganda is least likely to reach effectively. There are very few people who do not go to the pictures occasionally, and a suitably prepared "movie short" or even coloured lantern slides would bring home to those who allow dogs access to diseased offal the serious consequences of their neglect. I think the best way for me to deal with my subject will be to analyse the cases that it has fallen to my lot to deal with during the last eight years. Unfortunately, my records during part of this period are incomplete, but I have been able to trace the histories of fifty-nine infestations.

The incidence as it affected the various organs was as follows:

Liver	35 cases
Lung	16 cases
Pelvis	4 cases
Muscles	3 cases
Subcutaneous tissue	1 case
Spleen	1 case

I propose to deal with the various organs in turn, discussing briefly the pathology, diagnosis and problems of treatment of hydatid disease as it affects those organs. A few reports of cases are included when they seem helpful in illustrating problems in diagnosis and treatment.

THE LIVER.

The series of 35 liver cases emphasizes the predominance of right lobe involvement. The right lobe was involved in 23 cases, the left lobe in five cases, both lobes in three cases; in four cases the site of involvement was doubtful. Multiple cysts of the liver were found in three cases, but were probably present in more than that number.

The pathology of hydatid disease in the liver shows well how latent these cysts can be. The patient often does not seek advice until late adult life is reached, although even in such cases infestation probably occurred in childhood. Growth is slow, and until some complication occurs the effect is purely mechanical, if felt at all.

Age Incidence.

The ages at which the patients with liver infestations sought treatment is shown in Table I.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on November 21, 1936.

TABLE I.

Age Period.	Number of Cases.
Under 10 years	2
10 to 20 years	2
20 to 30 years	6
30 to 40 years	3
40 to 50 years	5
50 to 60 years	9
60 to 70 years	6
Over 70 years	2

Type of Cyst.

As was to be expected, the cysts in all the patients under thirty were of the simple univesicular type. The remainder had undergone some complication, usually biliary contamination following erosion of a bile duct, and contained daughter cysts of varying number and size, with, generally, a very degenerated laminated membrane.

Symptoms.

The symptoms for which the patients sought treatment are shown in Table II.

TABLE II.

Symptom or Group of Symptoms.	Number of Cases.
Pain and jaundice (intrabiliary rupture)	10
Symptoms probably due to anaphylaxis, giddiness, pallor, pain, urticaria, tight feeling in the chest, shock	7
Pain or discomfort	7
Presence of a tumour	3
Dyspepsia	3
Presence of a tumour and pain	3
Weak feeling	2

Complications.

The most important complications encountered in this series of cases were intrabiliary rupture, intraperitoneal rupture, suppuration and intrapleural rupture.

Intrabiliary Rupture.

Intrabiliary rupture occurred in ten cases. This is the most common complication; it is more common in this country than intraperitoneal rupture.

Pathology.—Owing to the resistance of the bile duct epithelium, biliary ducts became opened, leading to contamination of the cyst with leakage and reactive daughter cyst formation. Probably ten to twenty years later the cyst reached its original size and erosion of the wall of a large bile duct occurred. The contents of the cyst then escaped into the biliary passages, leading to temporary or persistent blocking by *débris* or daughter cysts.

In all these cases rupture occurred into the intrahepatic ducts, and in none was the cyst deeply situated in the centre of the liver. The offending cysts were situated on the superior or superior and lateral surface in four cases and on the anterior or antero-superior surface in five cases.

Age of Patients.—The youngest patient was aged twenty-nine years, one was thirty-eight, and eight were over fifty years, demonstrating very forcibly that frank intrabiliary rupture is usually a complication of old cysts.

Symptoms.—Biliary colic, but usually less severe than gall-stone colic, followed by jaundice occurred in all the cases. In one case attacks had recurred for a period of twelve years with intervals of varying length, the attacks being due to the intermittent passage of *débris* down the ducts.

In five cases severe infection followed, accompanied by rigors. Enlargement of the liver was present in every case, either upwards or downwards.

Diagnosis.—A correct pre-operative diagnosis was made in nine cases. The tenth case, which is described below under "Intrapleural Rupture", was not diagnosed during the life of the patient. In three the diagnosis was strongly suggested by a previous history of hydatid disease. In the other cases the diagnosis was made from the presence of hepatomegaly and the X ray findings (distortion and displacement upwards of the diaphragm), together with the result of the Casoni test. Examination of the stools for bile-stained membrane and daughter cyst is a most important and often conclusive method of investigation, but was not necessary for purposes of diagnosis in this series.

Treatment.—Direct attack on the offending cyst was the method of treatment adopted in every case. No deep cyst was responsible for the symptoms, and in no case was it necessary to deal directly with the ducts. The cyst was drained through a transcostal approach in four cases and through the anterior abdominal wall in five. Drainage persisted for some months in four cases owing to calcification of the adventitia.

When the site of the cyst requires a transcostal approach, the incision should be made as low down and as far forward as possible to avoid opening the pleural cavity. When this cannot be avoided, a two-stage procedure should be adopted, if at all possible. In a one-stage operation, however much care be taken in shutting off the pleural cavity, the risk of pyopneumothorax is a very real one. This occurred in one case, and, although the infection was not very severe, some months elapsed before cure of the resulting empyema took place. A report of the case is attached, because it is in other ways typical of the cases of frank intrabiliary rupture that occurred in this series.

E.S., aged twenty-nine years, complained of painful indigestion for two years, followed by wind after meals without actual pain. A week previous to his admission to hospital severe pain occurred in the upper part of the abdomen and under the right shoulder blade. Following this attack he felt ill and had shivers, with a temperature up to 40° C. (104° F.) at night. The skin became yellow and the urine dark coloured.

On examination the patient had a temperature of 39·4° C. (103° F.) and a firm, smooth, tender swelling in the epigastrium and right hypochondrium; the liver dulness was enlarged upwards and the urine contained bile. The Casoni test gave an immediate positive reaction. A right paramedian incision was made and a hydatid cyst was found presenting under the right dome of the diaphragm. The abdominal incision was closed and the cyst was opened

after rib resection in the mid-axillary line after careful isolation of the pleural cavity by suture and suture of the adventitia to the diaphragm. A large amount of bile-stained *débris* was evacuated, together with numerous daughter cysts of varying size, and a large drain was inserted.

The jaundice disappeared after ten days, but the temperature, after subsiding, began to rise again to 38·9° C. (102° F.). A pint of turbid fluid was removed from the right pleural cavity by aspiration on two occasions, followed by closed drainage after rib resection. However, expansion of the lung was very slow and there is still an occasional discharge after four years.

Results.—One patient died from suppuration in a second deep-seated cyst four years later; seven completely recovered and have remained well or have died from other diseases. One patient has had two subsequent operations, but has been well now for four years. One patient died from intrapleural rupture.

Intraperitoneal Rupture.

Intraperitoneal rupture occurred in two cases; in neither of them was hydatid disease suspected until rupture occurred.

The first patient ruptured a univesicular cyst on the superior surface of the liver as the result of a fall from a hay cart.

G.F., a male, aged nineteen years, was admitted to hospital in a state of shock, possibly in part due to anaphylaxis, and was operated on with a diagnosis of ruptured liver. A good deal of deeply blood-stained fluid escaped from the incision, and the gap in the upper surface of the liver, which could be felt but not seen, was plugged with gauze. Hydatid disease was not even suspected until the subsequent discharge of portion of the cyst wall following removal of the gauze occurred. The cyst wall gradually came away and the wound healed.

This happened four years ago. When last seen, the patient was well; but almost certainly extensive secondary cyst formation will occur and manifest itself in the next few years.

The second patient was referred as suffering from appendicitis.

D.B., a female, aged twenty-three years, gave a history of indefinite abdominal discomfort for six weeks. She had a tight feeling in the chest for a few days, and right lower abdominal pain for twenty-four hours before her admission to hospital. At operation a ruptured cyst was found projecting from the lower lateral part of the right lobe of the liver, together with a leaking cyst occupying the pelvis. The pelvic cyst had no adventitia, and laminated membrane was in direct contact with ileum, appendix, pelvic and abdominal wall.

In this case apparently extrusion of the intact mother cyst had occurred with comparatively mild anaphylactic symptoms. Choleperitoneum did not occur, because, as the cyst projected largely from the liver rather than being buried in its substance, no bile ducts had been eroded. Secondary echinococcosis from leakage of brood capsules and scolices from the cyst is almost certain to make itself evident in five to fifteen years.

Suppuration.

Suppuration with frank pus occurred in four cases. This complication is responsible for most of the deaths in hydatid disease, and in this series two patients died.

The nine cases complicated by intrabiliary rupture were associated with a greater or less degree of infection, but in only one case did actual abscess formation occur.

F.G., a male, aged fifty-six years, was treated by transthoracic drainage for a hydatid cyst of the dome of the liver with frank intrabiliary rupture and infection. The temperature subsided and jaundice disappeared soon after operation, but although the patient felt fairly well, the wound discharged intermittently until four years later, when he developed pain in the liver region and a temperature of 39·4° C. (103° F.).

Cœliotomy showed the epigastric tumour principally to be left lobe of the liver which had undergone compensatory hypertrophy. No hydatid cyst or abscess could be found, so the old sinus track in the axillary line was explored deeply in all directions through dense scar tissue. Eventually free bleeding necessitating plugging was encountered, but no pus was found.

Post mortem a large abscess was found with very thick walls deep in the liver substance. It could have been reached only by incising the liver substance deeply on its inferior surface by an intraperitoneal approach.

J.K., a male, aged sixty-five years, had no symptoms until a fortnight before his admission to hospital, when discomfort and swelling in the upper part of the abdomen, followed by vomiting, occurred. On examination a large, rounded, tender mass was found in the epigastrium, and under a local anæsthetic a thick-walled abscess containing 300 cubic centimetres (ten ounces) of thick pus was opened. Recovery ensued. The Casoni test gave a positive reaction, but the complement deviation test was not performed.

F.G., a male, aged fifty-five years, complained of loss of appetite and upper abdominal pain for three weeks. On examination he had a temperature of 39·1° C. (102·5° F.) and a large tender liver. The Casoni test gave a doubtful result and the complement deviation test gave no reaction. A large abscess with thick walls in the lower part of the right lobe of the liver was opened; it contained shreds of material which may have been degenerated hydatid membrane, but unfortunately they were not examined.

This patient gave no immunity reactions, but clinically the appearance of the abscess was similar to that seen in the other three cases, and no other cause for suppuration could be found.

The fourth patient came for treatment with a grave anæmia of an aplastic type, and hydatid disease was not suspected during life.

A.B., a male, aged thirty years, came complaining of two severe attacks of influenza during the previous twelve months. For the previous fortnight he had felt very weak. On examination pallor was very pronounced. The red cells count was 1,400,000 per cubic millimetre, the hæmoglobin value was 30%, and the white cell count 4,000 per cubic millimetre, 90% being lymphocytes. There were no other physical signs and the liver was not obviously enlarged. A fortnight later the temperature rose and continued at a fairly high level until the patient's death some weeks later, the degree of anæmia steadily progressing in spite of blood transfusion. No immunity tests were performed and no eosinophile cells were present in the blood film.

Post mortem examination at the Melbourne Hospital disclosed a suppurating hydatid cyst, three inches in diameter, lying between the right and left lobes of the liver. The cyst had thick walls and contained thick, yellow, foul pus. A *Bacillus coli communis* septicæmia with metastatic infection of the kidney and colon was present, together with an associated chronic peptic ulcer.

Intrapleural Rupture.

Intrapleural rupture occurred in two cases.

The first case occurred in a man of sixty-three, who had, a short time before, been operated on by the abdominal

route for intrabiliary rupture of an hepatic cyst. He recovered after a prolonged illness which necessitated a thoracoplasty.

The second case occurred in a male, aged fifty-nine years, who had been receiving compensation for lead poisoning for some years. He gave a history of attacks of abdominal pain and vomiting accompanied by jaundice for six months, but the cause was not suspected. He was very ill when admitted to hospital and died a few days later unoperated on. *Post mortem* examination revealed a suppurating cyst, the size of a big fist, which had burst through the diaphragm into the right pleural cavity.

THE LUNG.

Fifteen cases of hydatid of the lung and one of the pleura or extrapleural tissues occurred.

Thirteen of the patients were males and three females. The right lung was involved in ten cases, the upper lobe being affected in two and the lower in eight. The lower lobe of the left lung was affected in four cases and both lungs were affected in one.

One cyst occurring in a male, twelve years of age, and situated between the pleura and ribs in the posterior and lower part of the right thorax, is included as possibly primarily pleural in origin. The cyst was about 7·5 centimetres (three inches) in diameter and projected into the pleural cavity, being covered by parietal pleura, which was firmly attached to it only in a small part of its extent. There were no adhesions between the two layers of the pleura and no lung hydatid was present.

The ages at which the patients sought treatment are shown in Table III.

TABLE III.

Age.	Number of Cases.
11 years	1
12 years	1
13½ years	1
19 years	4
21 years	1
23 years	1
28 years	1
29 years	1
30 years	1
32 years	1
35 years	1
55 years	1
78 years	1

Type of Cyst.

All the cysts were univesicular, except one recurrent hydatid at the apex of the right lung, which contained a number of daughter cysts. According to the patient's statement, twenty-eight years elapsed between the two operations—some of the germinal cells had survived and developed later into daughter cysts. Daughter cyst formation is very unusual in pulmonary cysts, because rupture usually occurs before the cysts are very old, and this complication results usually in the expectoration of the cyst with natural cure or death of the parasite from sepsis.

Symptoms.

As in the case of liver hydatid, there are few, if any, symptoms before the onset of some complication. In this series three cysts were discovered during routine examination, and of these, two subsequently ruptured into bronchi. In six cases persistence of symptoms after respiratory infections led to the discovery of cysts; one of these subsequently ruptured into a bronchus. Three patients came with symptoms of intrapleural rupture. Two came complaining of pain. One came after intrabronchial rupture. One came complaining of a swelling at the site of an earlier operation.

K. D. Fairley has remarked on the fact that "a previous respiratory infection may be the starting point of symptoms which lead to the recognition of the cyst. Persistence of symptoms after acute respiratory infections should arouse suspicion of hydatid disease". The experience of these cases confirms this statement. Investigation of persisting symptoms led to the discovery of hydatid disease in four cases; in some of the cases with other complications an apparent pulmonary infection persisted until the calamity occurred.

Complications.

Rupture into the Pleural Cavity.

Rupture into the pleural cavity occurred in three cases. Two cases will be described in some detail, because they show well many of the points associated with hydatid disease of the lungs and the surgical problems these cases present.

F.N., a male, aged thirteen and a half years, gave a history of a cough ever since having had influenza six months previously. A week before admission to hospital he got a sudden severe cough and vomited up food and much phlegm. Since then he had had attacks of coughing and shortness of breath, with a temperature up to 39·4° C. (103° F.) and much foul expectorations.

Examination revealed a right pyopneumothorax with displacement of the heart to the left, and a positive immediate Casoni reaction.

On account of the extreme dyspnœa, an attempt was made to keep the pleural cavity empty by suction drainage through an intercostal space for a fortnight. This was unsuccessful, on account of the presence of a broncho-pleural fistula, and a thoracotomy was done with much difficulty on account of the severe dyspnœa, incessant cough and the impossibility of giving ether by the intratracheal route in the presence of a large bronchial fistula. The pleural cavity was cleaned of fluid and hydatid membrane and closed drainage was instituted. A month later there was still pyrexia up to 38·9° C. (102° F.) and very little evidence of obliteration of the pleural cavity, so the bronchial fistula was closed through a long intercostal incision. Two months later much improvement in the general condition had taken place and the cavity had become much smaller, owing to expansion of the lung, ascent of the diaphragm and collapse of the chest wall. Four months after the patient's admission to hospital the wound had closed and the patient was feeling well. A considerable degree of scoliosis had occurred owing to collapse of the thorax on the right side, and this was corrected by a plaster jacket cut away on the right side and the use of pads of increasing size between the jacket and chest wall on the opposite side.

This case shows well the danger of the yielding mediastinum of a child and the help that the flexibility of both the chest wall and the soft structures can give in the obliteration of the pleural cavity when the lung is slow in expanding.

The second patient was shown at a Branch meeting held at Camperdown in 1931; he then had a broncho-pleural fistula.

B.G., a male, aged twenty-nine years, was treated in the Coleraine Hospital a month previously with "fluid round the right lung", and there had been a tight feeling in the chest since then. Four days prior to his admission to hospital the patient had an attack of vomiting and pain in the joints (symptoms probably anaphylactic in nature).

The Casoni and complement deviation tests gave positive reactions. Physical examination revealed the presence of a hydropneumothorax. As infection did not occur and he seemed well, no active treatment was adopted. During this period several pieces of laminated membrane were coughed up.

Five months later, infection of the large residual cavity occurred, manifested by pyrexia and severe cough. Thoracotomy was done and a large, firm, walled cavity, containing degenerated hydatid membrane, was drained. An attempt, which proved unsuccessful, was made to close the bronchial fistula. The fever subsided and the external opening closed in three weeks, although the lung had not expanded and the bronchial fistula was still present.

The patient worked hard for fourteen months after this, and then reinfection of the cavity occurred after a cold. After some months of ill-health, in spite of drainage of the cavity, a Sauerbruch thoracoplasty was done in two stages. Satisfactory recovery ensued and the patient has been working hard for four years.

The difficulty in dealing with these two cases depended on the presence and persistence of broncho-pleural fistula, which prevented expansion of the lung and through which infection of the pleura occurred.

Rupture into a Bronchus.

Rupture into a bronchus occurred in four cases. In three of these infection occurred and operation was urgently required; the fourth patient expectorated the cyst and has since remained fairly well. In one case of bilateral pulmonary cysts almost simultaneous rupture and infection of both cysts occurred.

Sir Louis Barnett, writing in *The Australian and New Zealand Journal of Surgery* of January, 1936, states that the operative prognosis of cysts of both lungs is appalling, but in this case the outcome was a happy one.

This patient, a woman of thirty-five years, was extremely ill for a week after rupture of the large cyst (left lower lobe) at her home in the country and could not be moved for a week. Thoracotomy was then performed under local anæsthesia with removal of portion of the eighth rib posteriorly opposite an area where the two layers of the pleura were adherent. Much foul pus, together with degenerated membrane, was evacuated. Although the patient's general condition improved, the pyrexia continued, and it seemed probable that the second cyst was also infected. This was opened and drained after a juxta-sternal resection of the third right costal cartilage. Following this, rapid recovery ensued and both wounds were completely healed six weeks after the second operation.

One case was that of an old man of seventy-five years, who, after having had a severe cough following "influenza" twelve months previously, suddenly coughed and vomited up a very large amount of thick foul pus. He was so seriously dyspnœic that operation could be performed only with the patient sitting upon the edge of the operating table. He too made a satisfactory recovery so far as his pulmonary condition was concerned.

In these two cases the pleural layers were adherent, greatly reducing the dangers of operation. In the third case, although there were no pleural adhesions, the patient did well after a one-stage operation, but in most cases it is safer to deal with the infected cyst in two stages.

CYSTS OF MUSCLE.

Three cysts occurred in muscle, one in the psoas and two in the extensors of the thigh. The case of hydatid disease of the left psoas muscle occurred in a female, aged twenty-six years.

A cyst, 5·0 centimetres (two inches) in diameter, was found almost completely buried in the muscle fibres. Removal of the laminated membrane and closure after careful formalinization were followed by the development of secondary cysts or the continued growth of cysts formed by exogenous development and overlooked at the first operation.

At the second operation, two years later, a number of cysts of varying size were found attached to one another and burrowing in all directions—so soon as one cyst was dealt with, another was found deep to it. The patient is well at present, eighteen months after her second operation, but the future outlook is doubtful; unfortunately, it has not been possible to persuade her to have an X ray examination of her spine made to exclude a bony origin.

Two cases occurred of cysts in the extensor muscles of the thigh—both of these were primary and one of them contained daughter cysts.

CYSTS OF THE PELVIS.

Four cysts of the pelvis are included in the series.

A female patient, aged twenty-three years, had a cyst in the pelvis, apparently as the result of extrusion of the intact mother cyst after rupture of the adventitia in the liver. Later, leakage occurred, giving rise to lower abdominal pain, which was suspected of being appendiceal in origin.

A male, aged seventy-three years, had a large pelvic cyst, giving rise to an abdominal tumour, dysuria and constipation. No liver cyst was found, but there was a vague history of pulmonary hydatid many years previously.

A male, aged sixty-three years, came for treatment with a frank intrabiliary rupture of an hepatic cyst. On examination he was found to have as well a large cyst filling the pelvis, but giving rise to no symptoms.

A male, aged fifty years, had multiple cysts in his abdomen and pelvis following rupture of a liver cyst from a blow.

The first and fourth of these cases were obviously secondary, and it is impossible to say that the second and third cases did not originate from an unrecognized rupture of an hepatic cyst.

CYST OF THE SUBCUTANEOUS TISSUE.

One cyst occurred in the subcutaneous tissue over the right deltoid muscle in a female, aged nineteen years, who also had a large hepatic cyst.

INVESTIGATION AND DIAGNOSIS.

Many points in the diagnosis of hydatid disease have already been touched on in the reports of cases. In two cases of intraperitoneal rupture of hepatic cysts the diagnosis was made only during operation, and in one of deep-seated suppuration of an hepatic cyst the diagnosis was made only *post mortem*. In one case of cyst of muscle the diagnosis was made only at operation. In all the other cases a correct pre-operative diagnosis was made from the history and clinical examination aided by immunity tests and radiography.

Immunity Tests.

The Casoni test was done in 29 cases with the results that are set out in Table IV.

TABLE IV.

Site of Cyst.	Reaction Positive.	No Reaction.
Liver	15	1
Lung	6	4
Pelvis	1	

Three negative results were obtained in unruptured simple pulmonary cysts, one was obtained in a pulmonary cyst two days after intrabronchial rupture, and one in a large hepatic cyst complicated by intrabiliary rupture. In the first three cases absorption of antigen had not occurred, and in the other two cases presumably desensitization had taken place.

These results all refer to immediate reactions, the delayed reaction not having been recorded regularly. Hydatid fluid from the Walter and Eliza Hall Institute was used in every case, and the standards laid down by Fairley, Fairley and Williams in THE MEDICAL JOURNAL OF AUSTRALIA of September 7, 1929, were observed.

The complement fixation test was done in only five cases. There were two positive results, one in a case of intrapleural rupture of a pulmonary cyst, in which the result of the test suggested the diagnosis that had not been suspected before. The three negative results were obtained in: (i) a residual liver cyst in a case in which hydatid cysts of the liver and spleen had been operated on two years previously, (ii) an hepatic cyst containing thick pus, (iii) a large calcified hydatid cyst of the left lobe of the liver.

Radiography was by far the most important factor in the diagnosis of the presence and site of pulmonary cysts. In three cases in this series symptomless pulmonary cysts were recognized during routine screening of the chest. In cysts which have undergone calcification the use of radiography is also decisive.

ANAPHYLAXIS.

As is well known, many symptoms difficult of explanation in hydatid disease are probably due to the leakage of hydatid fluid in a patient already sensitized to the hydatid antigen. Such symptoms may be referable to any part of the body. Giddiness in several cases was the only symptom causing the patient to seek advice. Urticaria and pyrexia (the latter especially following operation) were among the commoner manifestations and occurred in a considerable proportion of the cases. Tightness in the chest and shock were associated with leakage in two instances. Only in one case did dangerous symptoms occur.

F.N., a female, aged sixty-four years, had been seen some months earlier with a hydatid cyst of the liver reaching down to the umbilicus. Some weeks later the liver was found to be a good deal smaller in size; evidently leakage into the bile ducts had occurred. Early one morning she suddenly became unconscious, with all the signs of severe anaphylactic shock. She was quite pulseless when first seen; she gradually rallied after an injection of one cubic centimetre of adrenaline (1 in 1,000), but was very ill for twenty-four hours. In a few days she was quite well again, but refused operation until five years later, when frank intrabiliary rupture with jaundice and infection occurred.

RESULTS.

There were six deaths. Three patients died from sepsis occurring in liver hydatid; two of them died unoperated on and with the condition undiagnosed. One patient with a pulmonary hydatid cyst died from pneumococcal meningitis a week after removal and closure of a cyst which had been complicated by pleurisy some weeks previously. A second died of tuberculous meningitis while still unoperated on, but this patient cannot be included as dying from hydatid disease. One patient with liver hydatid and secondary peritoneal echinococcosis died after numerous operations. One patient with hydatid of the liver died of pulmonary embolism three weeks after operation.

Of the remaining 53 patients, one is still awaiting operation. The others have been operated on and are well at present or have died from other diseases. The majority of those who are alive are probably cured, but several will doubtless need operations for secondary cysts in the future.

ACKNOWLEDGEMENT.

In conclusion I wish to express my indebtedness to Professor H. R. Dew, Bosch Professor of Surgery in the University of Sydney, for his ready help in any problems that have occurred in this series of cases.

HYDATID DISEASE IN CHILDREN.[1]

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A REVIEW of the case histories of the patients who have been investigated and treated for hydatid disease at the Children's Hospital, Melbourne, since 1900, will serve to bring out a number of points of interest; but it is not possible in the time at my disposal to cover the whole subject. Very meagre details are in existence for the first half of the period and this information has been used to supply the facts concerning the incidence and distribution of the cysts. Fairly full clinical information has been recorded at the hospital since 1920, and I have taken more than a passing interest in those children who have been admitted to the hospital with hydatid disease since then. I have been stimulated to do so by my friendship with such well known workers on hydatid disease as Dr. Charles Kellaway, Professor Harold Dew, Dr. N. Hamilton Fairley, Dr. Keith D. Fairley and Miss F. E. Williams, who have made the Walter and Eliza Hall Institute at the Royal Melbourne Hospital world-famous as a result of

[1] Read in abstract at a meeting of the Victorian Branch of the British Medical Association, November 21, 1936.

their publications of research work on this subject. The patients at the Children's Hospital have been under the care of many of the members of the honorary medical staff of the hospital and I wish to express my appreciation of the courtesy of my colleagues in allowing me to make use of the case histories of their patients.

Incidence.

Between July 1, 1900, and June 30, 1936, 151 children have been admitted to the hospital with hydatid disease. They represent 1 in 536 admissions for the period July 1, 1910, to June 30, 1921, and one in 1,240 admissions from July 1, 1921, to June 30, 1936. Reference to Table I will show that the absolute numbers of patients with hydatid disease are also decreasing. There was a yearly average of 7·4 patients in the first ten years of the period under review, but since 1930 the yearly average has fallen to 2·2 patients. To what extent these figures indicate a true decrease of the disease in the State of Victoria is dependent on the degree to which they are being treated in country centres where the hospitals have been able to undertake an ever-increasing amount of surgical work. It is almost certain, however, that the framing of by-laws to prevent access of dogs to offal at slaughter yards and the more rigorous inspection of meat and the widespread appreciation of the general public, especially in the rural districts, of the mode of spread of the disease have led to an actual diminution in its incidence. Another important factor in this connexion is the improvement in medical and surgical practice which has led to earlier and more certain diagnosis and more efficient treatment. The patients do not present themselves so frequently nowadays with recurrences.

TABLE I.
Annual Incidence at the Children's Hospital.

Period.	Number of Children.	Yearly Average.
1900 to 1910	74	7·4
1910 to 1920	36	3·6
1920 to 1930	28	2·8
1930 to 1936	13	2·2

It is of interest to note from reports in the literature that hydatid disease appears to be increasing in the United States of America.

Sites of the Initial Cysts.

The sites of the initial cysts present in the children admitted to the Children's Hospital between 1900 and 1936 have been tabulated (Table II). When multiple cysts were present in the liver or in the lung, they have been counted as liver or lung singly, but when cysts were present in each situation they have been listed under liver and lung.

The relative frequency with which cysts are found in children in the lung and in the brain by comparison with adults is probably accounted for by the probability that in such sites symptoms will show up earlier. It is generally recognized that a large proportion of all hydatid infestation takes place in childhood and that many cysts remain latent and undiscovered for a very long time.

TABLE II.
Sites of Initial Cysts in 151 Children.

Site.	Number of Cases.
Liver	87
Liver and lung	6
Lung	37
Brain	6
Abdominal wall	6
Pelvis, femur, rib	3
Peritoneum	2
Kidney	2
Spine	1
Orbit	1
Total	151

Major-General R. M. Downes,[1] in 1911, in a paper on pulmonary cysts in children, published the distribution of the sites of the cysts in the children at the Children's Hospital between 1904 and 1910 and by the addition of the case histories of 74 children I am able to bring the series up to June 30, 1936 (Table III). The 74 children referred to have in particular formed the basis for the present study.

TABLE III.
Sites of Initial Cysts in 114 Children.

Site.	1904 to 1910. (R.M.D.)	1910 to 1936. (H.B.G.)
Liver	25	40
Liver and lung	3	3
Lung	6	19
Brain	1	4
Elsewhere	5	8
Total	40	74

The Age Factor.

In Table IV will be found the age distribution of 74 patients admitted between 1910 and 1936. The age listed is the age at the time of the patient's admission for the first time. Twenty-two of the children were between six and seven and a half years of age at this time. The youngest child was two years and eleven months of age. Children cease to attend at the Children's Hospital at fourteen years of age.

TABLE IV.
Age at First Admission (74 Patients).

Age in Years.	Number of Children.
2 to 3	1
3 to 4	1
4 to 5	6
5 to 6	6
6 to 7	14
7 to 8	9
8 to 9	5
9 to 10	6
10 to 11	10
11 to 12	6
12 to 13	7
13 to 14	3
	74

Only twelve simple liver cysts were found among 36 children under the age of seven years and six months, and in Table V it will be seen that multiple liver cysts, pulmonary cysts and cysts both in the liver and the lung were more prevalent in the younger children.

TABLE V.
Age Factor and Initial Site (74 Children).

Initial Site.	Number of Children.		
	Under 6 Years.	Between 6 and 7½ Years.	Over 7½ Years.
Liver, single cyst	4	8	19
Liver, multiple	3	3	3
Liver and lung	2	1	0
Lung	4	6	9
Brain	1	1	2
Abdomen	0	0	3
Kidney	0	0	2
Orbit	0	1	0
Spine	0	1	0
Rib	0	1	0

Rupture and Suppuration.

Suppuration has been much commoner in this series than one would have expected. Consideration of the cases leaves little doubt that rupture, due either to trauma or to pressure into mucus-lined channels included in the adventitious wall or to a combination of these two factors, precedes and causes the infection. This confirms the view of Craig and Lee Brown, who stated[2] that infection and suppuration had been accepted as the cause of hydatid rupture, but that in their opinion it was more likely that the reverse of this was true. Dew, though he was referring to pulmonary cysts when he stated[3] that in all cases leakage from the cyst was an essential preliminary to suppuration, will doubtless subscribe to the wider application of this principle.

Daughter Cyst Formation.

Daughter cyst formation was noted to be present in only twelve of the children. One does not hesitate to agree with Dew[4] that daughter cyst formation must be regarded as an expression of the activity of the germinal cells of the parasite to ensure the carrying on of the species. The natural provision for continuing the life-cycle of the *Echinococcus granulosus* past the larval stage is the formation of scolices in the brood capsules of the cyst. In many of the cysts the scolices and brood capsules are not free in the fluid while the cyst is comparatively young. By the naked eye they may be overlooked, and, though they give the inner surface of the cyst a granular appearance, certain cysts containing them may have been described as sterile. Microscopic examination of a scraping of the inner surface of the cyst wall will reveal the true state of affairs. As the cyst matures, the scolices become free in the fluid and, when technical rupture occurs, they escape via the mucus-lined channels to the outside world and so have a chance of entering the definitive host. If, however, the life and function of the mother cyst and its free scolices are threatened by infection from the mucus-lined channels or the blood stream in the adventitious structure, daughter cyst formation takes place to produce new cysts with resistant walls and, inside them, new brood capsules produce another generation of scolices which have a greater chance of perpetuating the species. In the circumstances it is not surprising that, in a series of hydatid cysts in children, scolices are less frequently noted to be present and daughter cysts are uncommon by comparison with a series in adults.

Hydatid Infantilism.

In 1920 Dévé[5] reported five examples of hydatid infantilism. In the present series of children there was one child in particular who was very puny and who gained weight rapidly when a huge cyst was removed from the liver. This case raises interesting points concerning the rôle of the liver in nutrition and the rapidity with which a cyst may grow.

CASE I (57,739).—At the age of two and a half years this little girl had an illness, regarded as general peritonitis of unknown ætiology, which was followed by lobar pneumonia. After six weeks of colic, when the child was four years of age, single hydatid cysts were removed from the left and from the right lobes of the liver. She remained well for fifteen months under observation and then had a recurrence of colic. At that time the anterior edge of the liver was noted to be only two fingers' breadth below the costal margin, but, when the child was six years old, the liver edge was more than a hand's breadth below the costal border and a huge cyst, at least 20 centimetres (eight inches) in diameter, which occupied a very large portion of the liver from top to bottom, was removed. Before this operation the child weighed only 12·15 kilograms (twenty-seven pounds), but a few months later she weighed 26·1 kilograms (fifty-eight pounds).

Hydatid Diagnostic Tests.

The Casoni intradermal test has been of great value at the Children's Hospital since 1923, but, owing to the multiplicity of individual testers and lack of standardization of the fluid used for testing and of the results obtained, it is not possible to contribute anything of scientific value from the case records. The precipitin test has not been used extensively. On the other hand, all the hydatid complement fixation tests have been carried out at the Hall Research Institute, and I have been privileged to examine the laboratory books in which have been noted the exact nature of the antigen used and all the other essential data, such as the limits of positivity or negativity, in terms of minimal hæmolytic doses of complement (M.H.D.). In general, it may be stated that the sera have failed to fix the smallest amount of complement used when a hydatid cyst was present only if very little or no leakage of antigen had occurred from the cyst to the host. After operative interference, rupture, or the onset of infection, the amount of complement fixed very rapidly rose considerably, though to a varying extent, and often remained sufficiently great to justify a definitely positive report for many months, though in the absence of further cysts negative reports were ultimately obtained. It may be of interest to quote a case report to illustrate these remarks.

CASE II (33,446).—A female child, who had always been sallow, became feverish and had a large liver at the age of four years and three months. A year later she became very thin and had two definite attacks of jaundice. The liver was much larger and the stools were clay coloured. Before operation the serum fixed only one and a half minimal hæmolytic doses of complement, though the Casoni intradermal test yielded an immediate positive result.

At operation the liver was very tough, and, after aspiration of the cyst, the cyst wall was taken out whole. At aspiration one and a half pints of clear hydatid fluid were obtained first, followed by ten ounces of bile-stained fluid and ten ounces of blood-stained fluid. The cyst was a single one and was in the right lobe of the liver. Before operation the white cells numbered 6,900 per cubic millimetre, of which 1·5% were eosinophile cells (103·5); but, eighteen days after operation, the leucocyte count had risen to 52,200 cells per cubic millimetre, of which 8% were eosinophile cells (4,176). Two days after operation the result of the intradermal test was strongly positive; within one hour the reaction had spread over the whole arm and a generalized urticarial rash appeared over the trunk, face and other limbs, persisted for six hours and then subsided gradually. Ten days after operation 45 minimal hæmolytic doses of complement were fixed by the serum. The child did not make an uninterrupted recovery; she was feverish, the abdomen was distended, and the notch in the liver margin was 13·75 centimetres (five and a half inches) below the xiphisternum.

Another operation was performed six weeks after the first one; the liver was adherent to the parietal peritoneum and three and a half pints of bile-stained fluid under very great pressure were aspirated and free bleeding occurred when the track to the cavity was enlarged. Ten days later the serum fixed 45 minimal hæmolytic doses of complement. The child recovered rapidly and, eleven years later, has not reappeared at the hospital.

Prognosis.

With pulmonary and hepatic cysts the prognosis is good for the survival of the patient, even when rupture and suppuration have occurred, provided that the child is over four years of age. With cysts in other sites the age factor increases the likelihood of a cyst in a vital spot showing up in childhood, and there is usually some indication to modify the favourable prognosis when necessary. In Table VI data will be found to illustrate these conclusions. Apart from case number 22,018, in which a small hydatid cyst was found in the liver at autopsy examination of a child who died of rheumatic heart disease, there have been only nine deaths among the remaining seventy-three patients with hydatid disease under consideration. The age, the site and the explanatory remarks will be found in Table VI.

TABLE VI.
Hydatid Mortality. Nine Deaths in 73 Patients.

Serial Number.	Age in Years.	Site of Cyst.	Remarks.
9976	12	Kidney.	Operation severe, cyst large.
14996	3½	Liver.	Patient young, cyst large.
15721	9	Liver.	Congenital syphilis.
23848	6	Spine.	Laminectomy.
37466	6	Brain.	Long history, severe pressure.
45882	6	Brain.	Operation, cyst not removed.
58153	7	Liver, multiple.	Tuberculous hip disease.
59221	3	Lung, multiple.	Patient young.
63905	12	Brain.	Operation, cyst not removed.

CASE III (59,221).—A male child, two years and eleven months of age, the youngest in the series, had always been frail, but for two weeks before admission to hospital had been vomiting and coughing. By radiography it was established that a collapsed cyst was in the right lung, and that an undamaged cyst was in the left lung. At operation clear fluid was obtained from the cyst in the left lung, which was the size of a small orange, and the cyst wall was incompletely removed. After two weeks the heart and mediastinum were displaced to the left, partial pneumothorax and collapsed lung had developed on the left side and a pulmonary abscess on the right side. In another week large abscesses were present on both sides and a huge quantity of foul pus came from the wound. Ten days later the child nearly died on coughing up a great gush of foul pus, lingered three days and then died. At the autopsy it was seen that practically no obliteration of the cavity on the right side had occurred, and no cysts were found in the liver or in other parts of the body.

The cases of a brother and sister, the only examples in the present series of the infestation of two children in the same family, will be of interest to show how children can survive very severe infestation.

CASE IV (15,714).—A hydatid cyst was found in the right epigastrium of this female child when she was six years of age and, within four months, the swelling became tender and larger, and the child was jaundiced and nauseated. When she was six years and six months old, a simple uncovered cyst was removed from the left lobe of the liver, and she made an uneventful recovery. Eosinophile cells amounted to 9% of the leucocytes in a smear examined at the time.

Five years later she reappeared at the hospital because of pain and bulging at the site of the old scar, which had been progressive for one month. In other respects she seemed to be quite well. At operation another large simple cyst was removed completely from the left lobe of the liver. Though the fluid was described as turbid, the culture was sterile, and no hooklets or scolices were found in it. No daughter cysts were seen. The Casoni intradermal test gave no reaction, but the serum fixed at least six minimal hæmolytic doses of complement. She was afebrile throughout the illness and left hospital with the wound firmly healed seventeen days after operation.

Reexamined at the age of thirteen years, she had remained perfectly well and looked particularly well-nourished and healthy, but by all the variations of the fixation test at least six units of complement were fixed and, in addition, the precipitin reaction was very definite. She is now almost twenty-six years of age and is married. She has not had any recurrence yet, and has enjoyed very good health and played lots of sport.

CASE V (18,750).—The brother of 15,714 (Case IV), had at the age of nine years and three months what was regarded as pleurisy in the right side of the chest, which was aspirated on two occasions. Eleven months later he was admitted to the Children's Hospital in an orthopnœic and cyanotic condition. For two weeks he had been very ill with fever and frequent unproductive cough, and very large quantities of fluid had been removed from his chest, which filled up rapidly. The clinical finding of right-sided hydropneumothorax was confirmed radioscopically, but, on aspiration, 2,300 cubic centimetres of turbid yellow fluid were obtained, from which staphylococci and streptococci were cultured. Five days later 900 cubic centimetres of similar fluid were removed. The leucocytes were estimated to amount to 11,000 cells per cubic millimetre, of which only 0·5% were eosinophile cells. The evening temperature was as high as 39·4° C. (103° F.) before operation. Portions of the wall of a suppurated cyst were removed at operation, and fragments escaped from the wound at intervals. He was discharged from hospital 109 days after operation, but it was known that a cyst was also present in a central situation in the left lung. This fact was established by radiography.

At thirteen years of age he was readmitted to hospital because, two days earlier, he had had a rigor, and since then had been feverish, with pain in the middle of the left chest. His serum fixed six minimal hæmolytic doses of complement two days before operation, and, at the

operation, a suppurating hydatid cyst was removed from a posterior position in his left lung. Later in the day he became dyspnœic and had an hæmoptysis. No daughter cysts were present. The wound discharged for three days only, and after a week he was afebrile. He left hospital twenty-six days after operation. The Casoni reaction was positive, and the serum fixed at least six minimal hæmolytic doses of complement at that time.

Reexamined at fifteen years of age, he had not had any further trouble. The precipitin reaction was strongly positive, and not less than four units of complement were fixed by his serum by the Harrison method, not less than six units by the ice-box technique with hydatid fluid antigen, and even stronger reactions were obtained with the use of alcoholic extract of cholesterin, though the limit of positivity was not ascertained. He is now twenty-nine years of age, and has not been troubled with any illness whatever since he was in the hospital. He is married and is robust. He plays tennis and polo, and leads an active life in the country.

The prognostic value of the complement fixation test at an interval of more than one year after the operative removal of a hydatid cyst is still uncertain, though, in view of the conclusions of Fairley and Miss Williams[6] in 1935, we cannot be sure that sufficient time has yet elapsed to enable us to be confident that these last two patients are not still harbouring other cysts. In view of the importance of this matter another case may be quoted.

CASE VI (20,108).—A male patient, at the age of ten years, had what was regarded as double pneumonia, followed by persistent cough with expectoration, at times blood-streaked, and loss of weight. Six months later he was admitted to the Children's Hospital with right-sided hydropneumothorax. One hundred and eighty cubic centimetres of clear fluid were drawn off; the fluid did not contain scolices or hooklets. A month later a skiagram suggested the presence of a hydatid cyst at the base of the right lung, and eosinophile cells amounted to 19% of the leucocytes in a blood film. Through a rib-resection wound a subdiaphragmatic swelling was detected. The chest was closed off and a hydatid cyst was removed from the upper surface of the right lobe of the liver. It did not contain any daughter cysts. The wound was closed without drainage, and the patient left hospital nineteen days after operation in a rather anæmic and delicate condition.

Two years and five months later he came up for reexamination, and was in apparent good health. The serum fixed four units of complement, but the intradermic test was not done. After a further period of one year and seven months he was seen again, and was a strong, healthy, manual labourer. He had not had any symptoms indicative of further hydatid disease, nor was anything suggestive of the presence of another cyst found on careful physical examination, yet his serum fixed three and a half units of complement by the Harrison method and four units by the ice-box technique with hydatid fluid antigen, and three and a half and six units respectively with alcoholic extract of cholesterin as antigen. The precipitin reaction was weakly positive.

On the other hand, another case illustrates the more usual significance of the continued positivity of the complement fixation test.

CASE VII (26,674).—A male patient was admitted to the Children's Hospital at the age of nine years with the history that, three months earlier, he had fallen about two feet off a tree, and had immediately become very dyspnœic. That evening he had expectorated a small quantity of bright blood, and had complained of severe pain in the left side of the chest, especially on inspiration. A chronic cough and dyspnœa on the slightest exertion, with severe bouts of vomiting, had followed, and on the day of his admission to hospital, after a short paroxysm of coughing, he had expectorated large quantities of fluid sputum. Left hydropneumothorax was present. The fluid obtained on aspiration contained amorphous cellular *débris*, but no hooklets or scolices were found, and culture proved sterile. The patient's temperature, which had been normal, rose to 39·4° C. (103° F.) within twenty-four hours of aspiration, and an intermittent evening rise of temperature persisted. The serum fixed not less than six units of complement.

A week later, after a violent bout of coughing, he expectorated a quantity of yellowish-green fluid, from which Type I pneumococci were recovered, but which did not contain any recognizable hydatid material or tubercle bacilli. Next day, under local anæsthesia, an intercostal incision was made, and from it there escaped fragments of a large cyst wall and a quantity of pus. No daughter cysts were present, nor could any hooklets be found, but numerous bodies, taken to be degenerated scolices, were seen, though no definite rostellum of hooklets could be identified. Free drainage was established, and the temperature quickly subsided. Two weeks afterwards, through a rib resection wound, a further large portion of cyst wall was removed. Convalescence was uneventful, and a month later the patient was discharged from hospital. Reexamined a year later, he had had no further symptoms, and no evidence of the presence of another cyst was obtained on thorough physical examination or even by radioscopy, but the serum fixed two units of complement with hydatid fluid antigen and the Harrison technique and four units by the ice-box method; with alcoholic extract of cholesterin as antigen six units were fixed with the Harrison and eight with the ice-box technique.

All went well for two years longer, when, at the age of twelve years and five months, the patient was readmitted to hospital with toxæmic symptoms and expectoration. A large cyst was located towards the apex of the upper lobe of the left lung, but, before operation, he became very dyspnœic and was almost asphyxiated when swallowing food. At the operation, a week later, a cyst about the size of a tennis ball was removed from the back after reflecting the scapula outwards. In the following week fifteen cubic centimetres of pus were drawn off from the apical portion, and, in another week, this area was opened and a large abscess cavity which contained three daughter cysts was emptied and drained.

A year later the boy was terribly emaciated and very pale, and over 200 cubic centimetres of purulent material were removed from the abscess cavity. Three months afterwards the infection had settled down and he appeared to be well. When he was fourteen years and four months of age he was reexamined, three weeks after hæmoptysis. Chronic cough had persisted, and his condition was poor, and pus discharged through the seventh left interspace. Skiagrams failed to show any further cysts.

Pulmonary Hydatids of Children.

Twenty-two out of the seventy-four children in the present series had pulmonary cysts, and the initial cysts occurred equally in the two lungs. This was a surprising finding, for it was thought that they would be much commoner in the base of the right lung than elsewhere. The reason for the relative frequency of pulmonary cysts in childhood as opposed to later life is a point of interest. It may be that the large liver of infancy is more readily threaded by hexacanth embryos than is the mature liver, but it is more likely that pulmonary infestation will manifest itself earlier in life than will hepatic infestation; and as the vast majority of infestations take place in childhood, the proportion of pulmonary cysts to hepatic cysts becoming manifest in childhood will be higher than the proportion in later life. Even in this series of children it can be shown that the pulmonary cysts were of clinical importance at an earlier average age than were the hepatic cysts. For the whole series the average age

at the time of the patient's first admission to the hospital was 8·4 years; if the children with pulmonary cysts are excluded, this average age for cysts elsewhere was 8·9 years. The average age on first admission of the patients with pulmonary hydatid was 7·4 years, so on the average the patients with pulmonary cysts came under notice eighteen months earlier in life than those with cysts elsewhere.

Apart from the simple pulmonary cyst which may be discovered accidentally by the detection of relative dulness or in a skiagram, a number of interesting clinical types may be separated, which include those that simulate bronchiectasis, empyema or phthisis. There is a distinct liability of pulmonary cysts to discharge into the mucus-lined channels on which they impinge, and suppuration is by no means uncommon. A few case histories will serve to illustrate these features.

CASE VIII (43,955).—This case is of the bronchiectasis type. A little boy had generalized urticaria at the age of four years and one month, and eight months later had an attack of pneumonia at the base of the left lung. Cough persisted until he had pneumonia again, in the same situation, at the age of five years and three months. Immediately after this attack he coughed up about one pint of dark blood and matter; his breath was very foul and his sputum frothy, but he was not very ill. Hydatid membrane was identified in the sputum. The base of the left lung was dull, with diminution of breath sounds and faint, almost cavernous breathing and showers of crepitations were audible there on coughing. The Casoni intradermal test gave a strongly positive result. The temperature subsided in a week and the child was not very ill throughout the febrile course. Hydatid laminated membrane was expectorated while the child was in hospital and apparently by this means spontaneous cure resulted. No operation was carried out, but the boy recovered quite satisfactorily, and eight years later had not reappeared at the hospital.

CASE IX (42,843).—This case is of the phthisis type. A boy was admitted to hospital at the age of nine years and six months. Three months earlier he had an attack of left basal pneumonia, during which hæmorrhage from the lung had occurred, and in the interval he had repeated hæmoptyses with cough and yellow sputum. No tubercle bacilli could be found in the sputum. On his admission to hospital the base of the left lung was dull and the dulness extended up to the middle of the scapula and out to the axilla, with diminished breath sounds and occasional crepitations. The liver was not enlarged. He looked healthy and felt well in spite of the history and the physical signs, and the hydatid fixation test yielded a triple positive result. At operation a dead suppurated cyst was removed piecemeal, but complete with contents, through a wound made by resecting a portion of the ninth rib on the left side. Recovery was complete in three weeks, and eight years later the patient had not returned to the hospital.

CASE X (34,210).—This case is of the empyema type. At the age of three years and eleven months a little boy had an attack of left-sided pneumonia with generalized urticaria and a recurrence of the pneumonia six weeks later. Again after an interval of six months he had pneumonia severely in the same situation and did not recover satisfactorily.

Two months later, at the age of four years and nine months, he was admitted for the first time to the Children's Hospital. Dulness with diminished breath sounds over the left upper lobe, elevation of temperature to 38·3° C. (101° F.) and a respiratory rate of fifty breaths per minute were the chief findings. The Casoni intradermal test gave an immediate strongly positive reaction and the hydatid complement fixation test a triple positive reaction. Eosinophile cells represented 2% of a total white cell count of 52,000 per cubic millimetre (1,040). At operation rather more than 300 cubic centimetres of yellow offensive pus were removed from a huge collapsed cyst, which was in the upper lobe of the left lung and not in the pleural cavity. The cyst wall was removed completely. The exploring needle was blocked on several occasions by minute cysts, which were regarded by Dr. Reginald Webster, pathologist to the hospital, as small daughter cysts. Hooklets and degenerated scolices were present in the fluid, from which were cultured *Staphylococcus aureus* and Friedländer's bacillus. Six weeks later the boy went to his home after a complete recovery. On reexamination at the age of five years and six months, he had had no further trouble, and the hydatid complement fixation was reported as a double positive with ice-box technique and a little less strongly positive with the Harrison method, but ten years later he had not reappeared at the hospital.

The characteristic circumstances in which daughter cysts formed, the early stage at which they were observed and their presence in a pulmonary cyst are features of special interest.

Cysts in the Abdomen, Kidney, Orbit and Vertebral Spine.

It may be of value to place on record the occurrence of cysts in unusual situations in the series, and with this object some more case histories are epitomized.

CASE XI (33,720).—This is a case of multiple abdominal cysts. At the age of three years a girl had severe constipation requiring the frequent use of enemata and strong aperients. Constipation remained a clinical feature, and she had two or three severe attacks annually, associated with abdominal pain and requiring medical attention. At the age of ten years and nine months, she was admitted to the hospital for investigation during one of these attacks. Before operation a very definite positive and almost immediate Casoni reaction was obtained; and by the Harrison method 30, by the ice-box technique 35 minimal hæmolytic doses of complement were fixed by her serum. Eosinophile cells formed 4·5% of the total leucocyte count of 24,600 cells per cubic millimetre (1,107). At laparotomy free fluid was found which was turbid, though sterile on ascitic agar. The fluid was loaded with red blood cells and polymorphonuclear leucocytes, and many hydatid scolices were found in it. Several cysts in the omentum and a large cyst in the broad ligament were removed intact. They were typical hydatid cysts; two of them contained small daughter cysts, and the fluid from each was particularly rich in scolices. No cysts were found in the liver and there was nothing to indicate that a liver cyst had ruptured, though it would seem probable that this had occurred a long time before. On the day after the operation eosinophile cells amounted to 29%, with a total white cell count of 23,000 cells per cubic millimetre (6,670).

The implantation and development of so few of the scolices in this case and the absence of any readmission of the patient in the subsequent three years are of special interest. She made a rapid recovery and left hospital two weeks after the operation.

CASE XII (69,605).—This is a case of cyst in the right kidney. At the age of eleven years a boy had an illness with vomiting, pain in the right iliac fossa and low, swinging temperature for three weeks with intermissions. Under general anæsthesia a mass was felt in the right hypochondrium. The urine contained pus cells and motile bacilli. The Casoni intradermal test gave no reaction. At operation a large cyst was found in the upper part of the right kidney; it had a smooth surface and was slightly fluctuant, and was fixed very strongly to all the organs in normal relationship with the kidney; the duodenum and transverse colon were so firmly adherent

to the mass that they could not be stripped off. Removal of the kidney presented great difficulty, so a large needle was plunged in and creamy yellow fluid was obtained; a lot of this fluid, with hydatid elements, including laminated membrane and various daughter cysts, escaped when the tumour was incised after the abdomen had been carefully packed off. A small diverticulum, which was merely an outlying part of the cyst itself, ran towards the duodenum. Three months later a number of small cysts and three large pieces of cyst wall were obtained on opening up a small sinus which had persisted. At this time a triple positive result to the complement fixation test was reported. Later still, the child passed several small daughter cysts *per urethram*.

CASE XIII (21,015).—This case was a cyst of the right orbit. At the age of six years and nine months a little boy was admitted to the hospital because of progressive protrusion of the right eye during the previous four months. The protrusion had become so extreme that the eye could be extruded from the orbital cavity and the lids fell behind it. The intradermal test gave a positive reaction and the serum fixed one and a half units of complement by the Harrison method before operation. Kronlein's operation was performed; the lateral wall of the orbit was turned out subperiosteally and the tumour was found lying between the lateral rectus muscle and the optic nerve behind the globus. During manipulation the tumour burst; fluid escaped and a definite cyst wall was taken away. A week later the serum fixed eight units of complement, and during the next week eosinophile cells formed 5% of the white cells and, later still, 15·5% with the total count of 21,250 leucocytes (3,294). The boy had definite weakness of the right external rectus muscle, but made a good recovery. Two months after the operation the serum fixed only two and a half minimal hæmolytic doses of complement.

CASE XIV (23,848).—In this case cysts of the vertebral spine were present. A little boy, at the age of six years and six months, was admitted to hospital because of pain in the left axilla and between and below the scapulæ two weeks after a bigger boy had knocked him over and sat on his back. A week later he had been unable to walk, and on admission an incomplete transverse lesion of the spinal cord had become manifest; this lesion rapidly became complete during the next two weeks. Skiagrams of the spine and examinations of the cerebro-spinal fluid failed to reveal any abnormality. The blood and cerebro-spinal fluid were examined by the Wassermann test with negative results. Spastic paralysis was followed by flaccid paralysis. Ascending anæsthesia and hyperalgesia reached a level regarded as that of the zones of the fifth to ninth thoracic segments of the cord.

Laminectomy was carried out seven weeks after the onset of the pain. The laminæ of the fifth to eighth thoracic vertebræ inclusive were removed. The cord looked blue and was non-pulsatile, but no cause was found. The boy died a few hours after the operation, and at autopsy thirty to forty small cysts were found on the right side under the second, third and fourth laminæ, and on the left side under the fourth and almost down to the fifth lamina, compressing the cord. The cysts were small hydatid cysts and no mother cyst wall could be seen.

Brain Cysts.

Brain cysts are so much more prevalent in childhood than in later life that no apology is required for elaborating the clinical facts in connexion with the four examples in the present series. The argument already advanced for pulmonary cysts applies with greater force to explain the relative frequency of intracranial cysts in childhood. Indeed it is remarkable that a cyst could be present for any length of time in this situation without giving rise to symptoms. A few comments are offered after the synopsis of each of these cases, and the synopses will serve to illustrate many of the clinical features of hydatid cysts in the head.

CASE XV (63,905).—A girl, at the age of twelve years, showed signs of increased intracranial pressure, and a subtemporal decompression operation was done on the right side. It was noted at the time that the child's mother had had hydatids of the pelvis removed on two occasions; the first of these operations was either just before or in the early portion of the pregnancy which resulted in the birth of the patient, and the other took place five years later.

A month after the subtemporal decompression the child was readmitted to hospital. Though she was well nourished, she was not walking as well as she had been; she had persistent pain over the right eye and twitching all over the body; no further vomiting had occurred, but she had been unable to feed herself for several days; cerebral herniation, which was pulsating and hyperæsthetic, had occurred at the site of the decompression opening, and she had right-sided facial paresis and paresis of the left leg with slight ataxia of the left arm and leg.

An attempt was made by ventriculography to throw some light on the position and extent of the tumour or cyst, but the results were disappointing and, as the girl got a severe pain in the neck and was shocked, the procedure was not completed. In one skiagram taken at the time bone erosion can be detected, but, as the report was directed to the interpretation of the ventriculogram, this fact was unfortunately overlooked at the time. The child died two months later, and her condition in the interval precluded the possibility of further operation, which was arranged and postponed several times.

At autopsy a large hydatid cyst was found immediately under the vertex; it was extradural, between the bone and the dura, compressing the brain and the sagittal sinus; extensive erosion of the bone with craniotabes on the one side was separated by the cyst from the frontal lobes, which were both greatly compressed on the other side. No daughter cysts were present, but a few scolices and detached hooklets were found in a centrifuged specimen of the fluid from the cyst. No other cysts were found anywhere in the body, and there was no indication that the cyst had been extruded from the brain.

In view of the doubt which has been expressed about the occurrence of extracerebral hydatid cysts, it may be of interest to state that Dr. Reginald Webster, who conducted the *post mortem* examination with his usual meticulous care, is certain that this cyst was extradural, and specimens both of the skull, with cyst wall attached, and the brain, showing the compression of the frontal lobes, have been mounted and may be seen in the pathological museum at the hospital.

This cyst could have been reached and removed if an opening had been made near the vertex of the skull. It is most regrettable that this attempt was not made while there was still time. The question is raised as to the diagnostic significance of the cerebral herniation. As will be shown later, intracerebral cysts seem to replace brain substance without causing greatly increased intracranial pressure. They may not even cause enough bulging of the dura into the wound to make certain that the surgeon will go on and find them.

CASE XVI (45,882).—At the age of six years a child had a febrile illness, which at first was thought to be influenza, but the presence of papillœdema was noted and bilateral paralysis of the sixth cranial nerve appeared but cleared up quickly. A month later the papillœdema was still present and the squint recurred; headache and vomiting became features. Right temporal decompression was done with some relief to the papillœdema, but there was no bulging of the membrane into the trephine opening or cerebral herniation.

Three months later, on reexamination, the papillœdema was worse and further decompression was under con-

sideration. In another two weeks, after earache, a brownish discharge came from the right ear and a suboccipital decompression was undertaken. The child died a fortnight later from septic basal meningitis, and at the autopsy a hydatid cyst the size of a small orange was found in the right frontal lobe, completely covered by cerebral cortex. No adventitious condensation could be identified, and no daughter cysts were found, but numerous scolices were present. No other hydatid cysts were found after a systematic search through the body.

We have in this case another example of the confusion of a frontal tumour or cyst with a subtentorial one; it is submitted that the signs of gross increase of intracranial pressure should not be expected in the case of an intracerebral hydatid cyst.

CASE XVII (37,466).—A little girl, at the age of five years and eleven months, had, for two years, been having headaches and vomiting, and had lost visual acuity, memory and strength. Post-neuritic atrophy of the right disk was reported on examination of the fundi. Slowing of the speech, loss of sphincteric control and ataxia passing to spastic weakness were the main features. The condition had affected the left arm and leg to a greater degree and in advance of the right limbs. The back and neck muscles were powerless, and the child was in a semicomatose state. Only a few drops of cerebro-spinal fluid were obtained by lumbar puncture. At operation in the right temporal region there was a gush of fluid on incising the temporal muscle, and the bone was found to be thinned like eggshell, and the *dura mater* was very thin. It was thought that the fluid was cerebro-spinal fluid and that internal hydrocephalus was present.

The child died soon after operation, and at the autopsy it was found that the right temporal region was the only portion of the skull that was thinned; a large hydatid cyst protruded onto the ventral surface of the right temporal lobe and offered no resistance to being picked out by forceps; the cyst was the size of a large orange, no formation of any adventitia could be found, and there was no dilatation of the ventricles; but in the right cerebral hemisphere the excavation produced by the cyst communicated with the posterior horn of the lateral ventricle in the occipital lobe. The nature of the parasite was established by the demonstration of laminated membrane and the presence of very numerous scolices, but a specimen of the fluid obtained at the operation was examined at the Walter and Eliza Hall Research Institute, and failed to yield a positive hydatid complement fixation reaction.

In contradistinction to Case XV, this cyst was extruded from the brain, and caused pressure atrophy of the bone of the skull at the site of contact. It is surprising that the condition was so advanced before the child was submitted to operation. No cysts were found elsewhere in the body, and, as in the previous cases, the cyst must be regarded as a primary one.

CASE XVIII (68,187).—A boy was admitted to the Children's Hospital in November, 1935, at the age of ten years. Severe convulsions had occurred on three occasions, three years, two years and three weeks, respectively, prior to admission. They lasted four hours approximately, and ceased only on the administration of chloroform anæsthesia. Serious deterioration of his mentality had occurred overnight in association with the most recent convulsion. Headaches had troubled him for a year, and the eyesight had been definitely defective for six months. On the left side there was general narrowing of the field of vision, and secondary atrophy of the optic nerve head was present; right homonymous hemianopia was also reported. The skiagram of the skull showed separation of the sutures; the Casoni test gave no reaction, but the result of the hydatid complement fixation test was reported as double positive by the Harrison method and triple positive with the ice-box technique. Eosinophile cells amounted to 10% of the total white count of 14,000 per cubic millimetre (1,400).

At operation the left parietal area was exposed, and the dura was found to be under tension, and a few fine adhesions were present. A resistance to probing was encountered at a depth of 3·5 centimetres immediately cranial to the mastoid region. The intervening brain was incised with a diathermy knife, and a large cavity was entered, from which fluid gushed. A cyst wall was removed, the cavity was swabbed with saline solution, the dura was sutured and the head closed. The temperature rose later that day to 40° C. (104° F.), and next day the maximum temperature reached 38·9° C. (102° F.). The boy's condition was desperate, and it was only after he had been given a transfusion of twelve ounces of blood that he regained consciousness. The cyst membrane was typically laminated and numerous scolices were present. The boy recovered, and was discharged from hospital twenty-five days after operation.

Reexamined nine months later, the child seemed to be well, though he was rather dull.

It is regarded as possible that he has a cyst in his liver, and his future progress will be followed. It is realized that a period of about five years must be allowed to elapse before we can feel sure that secondary cysts are not present in the skull. The diagnostic value of the complement fixation test for the presence of further cysts will be of great importance to this patient.

Summary.

1. The records of the Children's Hospital, Melbourne, from the beginning of the present century to June 30, 1936, have been searched for clinical material concerning hydatid disease in children. During this period one hundred and fifty-one children have been admitted to the hospital with hydatid disease. The histories of seventy-four children admitted since July 1, 1910, have formed the basis for the present study.

2. There has been a steady decrease in the incidence of the disease at the hospital, and in all probability throughout the State of Victoria.

3. The sites of the initial cysts present are tabulated, and the importance of the age factor on the distribution of the cysts has been examined and discussed.

4. The view that rupture of the cyst precedes and causes the infection is supported, and the conditions which determine the formation of daughter cysts are examined from the clinical aspect.

5. An attempt is made to indicate the relative value of the diagnostic tests used in the present series. The outstanding value of radiography has been taken for granted.

6. Prognosis is studied on the basis of the morbidity and mortality revealed in the series and illustrated by selected case histories.

7. Special reference is made to pulmonary cysts and brain cysts rather than to hepatic cysts, because of the relative importance of the former sites in childhood, and a series of case histories is included to show the clinical features of infestation in unusual sites.

References.

(1) R. M. Downes: "A Brief Review of Cases of Pulmonary Hydatid Disease Treated at the Children's Hospital During the Years 1904-1910", *Australian Medical Journal*, Volume XVI, March 23, 1911, page 127.
(2) R. G. Craig and R. K. L. Brown: "Hydatid Disease of the Kidney", *Surgery, Gynecology and Obstetrics*, Volume XLVI, May, 1928, page 668.
(3) H. R. Dew: "Hydatid Disease", 1928, page 323.
(4) *Ibidem*, page 65.
(5) F. Dévé: Quoted by H. W. Mills: "Hydatid Cysts in Children, with a Report of Three Cases", *Surgery, Gynecology and Obstetrics*, Volume XLII, May, 1926, page 592.
(6) K. D. Fairley and F. E. Williams: "The Complement Fixation Test in Hydatid Disease", *Royal Melbourne Hospital Clinical Reports*, Volume VI, December, 1935, page 81.

Reports of Cases.

PARALYSIS FOLLOWING INGESTION OF TRI-ORTHO-CRESYL PHOSPHATE.

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Clinical History.

K.W., A MALE, aged nineteen years, was first seen on June 6, 1934. The medical history of his childhood had been uneventful. During the preceding four years he had excelled at athletics, football and cross-country running. During the months preceding May 14, 1934, he had been employed by a firm which distributed chemicals used in industry. On May 14, in the course of his duties, he had to transfer several gallons of tri-ortho-cresyl phosphate from a bulk container into gallon jars. In order to start the flow of liquid from the bulk container, he applied his lips to the end of a length of rubber tubing attached thereto and aspirated some fluid into his mouth. As the liquid is heavy, this action was repeated some nineteen times during the afternoon.

On the evening following he felt and looked very ill, vomited several times and had several convulsions. His lips were sore; he complained of cramps, but had no neck stiffness. He improved slowly during the following week, but when walking was attempted on May 26 he was giddy and shaky and needed the support of two sticks.

On May 29 he went for a holiday to the seaside and attempted further walking. On June 2 he felt stiff and noticed definite weakness in the lower extremities. On June 5 he attempted to resume work, but could not move quickly and fell over.

The examination made on June 6 revealed bilateral symmetrical motor paralysis of both upper and lower limbs, affecting especially the muscles below the elbows and knees. No definite loss of sensation was detected.

During the next two weeks the degree of paralysis increased, especially in the intrinsic muscles of the hands, so that by June 23 the picture was at its worst. The muscles of both shoulders and elbows were normal; the extrinsic muscles of the wrist and fingers could act against gravity, but not against resistance; the intrinsic muscles of the hands were graded as poor to fair—not able to act against gravity. In the lower limbs the muscles of the hips were not affected. On both sides the quadriceps muscle could act against gravity, but not against normal resistance. There appeared to be no power of voluntary contraction of the calf muscles nor of the plantar and dorsiflexor or intrinsic muscles of the feet.

After the first examination, plaster moulds had been applied to hold the feet at rest in the neutral position and to rest the quadriceps. Hand splints made of "Cellona" had been applied to hold both hands in the position which would protect from stretching the intrinsic muscles—lumbricales and interossei and the thenar muscles—while holding the wrists in dorsiflexion.

Physical therapy was given by Miss Stella Andrew. At first it was necessary to use faradic stimulation. At the end of August, three and a half months from the onset, the extrinsic muscles of the forearms had recovered and the calf muscles of both legs showed a slight return of power of voluntary contraction.

By January, 1935, seven months from the onset, the intrinsic muscles of the hands had recovered sufficiently to allow freedom from splinting for several hours each day. Gradually during 1935 power returned to all affected muscles, so that by November, 1935, eighteen months from the onset, the patient was able to resume his work. At present, though able to walk normally and to carry on his work, he has not yet recovered the power he had previously developed by athletics, but improvement is still occurring.

Comment.

This case is of interest because of the ætiology. The orthophosphate of tri-cresyl is a bland oily non-volatile substance which looks like mineral oil, and has been used extensively in industry as a plasticizer. It has been employed mainly in the manufacture of celluloid and allied products, in the tanning of leather and in making paints and varnishes. The toxicity of this substance was not appreciated by the firm employing the patient, though its power to produce paralysis had been demonstrated dramatically in the United States of America during the years following the repeal of prohibition. Tri-ortho-cresyl phosphate was the chemical responsible for several thousand cases of paralysis of the muscles of the hands and feet described in American literature as instances of poisoning by Jamaica ginger and popularly known as cases of "Jake paralysis".

Jamaica ginger extract had been for many years a popular beverage. In 1929 it was discovered that the substitution of the orthophosphate of tri-cresyl for some of the oleo resin of ginger could produce a drink which resembled in taste the old Jamaica ginger extract, and which could undersell it. Since the discovery of the toxicity of this substance in America, the non-toxic isomeric meta- and paraphosphates of tri-cresyl have been substituted for the ortho-phosphate in industry.

The clinical picture presented by K.W. was typical of "Jake paralysis" as seen by me in the eastern States of America during 1932 and 1933, and as described in the extensive medical literature. The following summary of "Jake paralysis" has been extracted from an article by John G. Kidd and Orthello R. Langworthy, published in the *Bulletin of the Johns Hopkins Hospital* in 1933:

> The symptoms caused by the ingestion of tri-ortho-cresyl phosphate present a clear-cut clinical entity showing the following salient features:
>
> 1. The history of ingestion of some compound containing tri-ortho-cresyl phosphate.
> 2. A preparalytic stage of two to three weeks following the ingestion of this substance is succeeded by numbness and cramping pains in the calves.
> 3. Paralysis then develops in the muscles of the legs below the knees.
> 4. About one week after the onset of paralysis in the legs weakness develops in the muscles of the hands and forearms.
> 5. The paralysis is bilateral and affects both sides almost equally.
> 6. Fatal cases form a very small percentage of the total.
> 7. *Post mortem* examinations have demonstrated chromatolysis of the anterior horn cells and degenerative changes in the peripheral motor nerves; the tracts in the central nervous system being unaffected.
> 8. Paralysis can be induced in experimental animals—rabbits, chickens, dogs, cats and monkeys—by ingestion or injection of tri-cresyl orthophosphate.

Many cases of paralysis occur in man in which the ætiology cannot be determined. In the present complex civilization, poisons lurk in preparations the toxicity of which is not suspected. In view of the definite proof that this substance can produce paralysis, it is suggested that its introduction into industrial chemistry in Australia may produce other instances of paralysis unless its dangers are appreciated.

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE AUSTRALIAN BRANCHES: COMMON PROBLEMS AND MUTUAL UNDERSTANDING.

THE Australian Branches of the British Medical Association have many problems in common. As Branches of the Parent Body they have as their first care, of course, the study of the medical sciences and their off-shoots. In some parts of the Commonwealth what may be called regional diseases are seen—for example, in the northern parts of Queensland medical practitioners encounter conditions that are practically unknown in the southern parts of the continent. But on the whole the scientific problems of Australian practitioners are the same as those in other parts of the English-speaking world. The members of the Branches have many facilities for the study of the problems of medical science. They can buy books by the dozen on every system of the body and on almost every disease. They have journals of general and special interest, in which their own views and those of others may be expressed. They attend Branch meetings for discussion of scientific subjects and occasionally at these meetings speakers from other States and even from other countries may be heard. In matters medico-political discussion is not so general, nor are there many opportunities for the members of one Branch to learn much of the difficulties, methods of working and achievements of another.

A casual observer might think that friendly society lodge practice, workers' compensation matters, hospital practice and other aspects of medical life would be the same in all the States of the Commonwealth. This view would, of course, be very far from the truth. Conditions of life vary in different parts of the Commonwealth; different governments adopt different attitudes towards health problems, and sometimes strange legislation is enacted; the temper and temperament of friendly society members, of hospital boards and of workers are by no means similar in all parts of Australia; and—this is often forgotten—medical practitioners in one State may have a way of looking at their problems that is not easily understood by those in another. There is also sometimes in members of one Branch a strange unwillingness to try to understand the difficulties that face a group of members of their own Branch or those of another. Even in serious conclaves where mutual help and mutual understanding should be forthcoming, this has been evident. Apart from the fact that understanding and sympathy should be shown, even if help cannot be given, it should surely be remembered that the difficulties facing one group or one centre today may be those of the whole profession tomorrow.

Much the same can be said of medical communities in any one State. A medical community is just like any other group of people—the same cliques, friendships and prejudices are found, and in these circumstances it may be difficult for the whole body to seek the common good. Matters of serious import are best handled by the Council of the Branch of the British Medical Association in the particular State. It is quite conceivable that a group of practitioners associated for a particular object, holding certain appointments or charged with certain responsibilities may feel disposed to

take an action that in the long run will embarrass the whole profession in the State. In these circumstances it will be wise to discuss the proposed action with the only body that is representative of the whole profession. The councils of the Branches are generally well informed on the medical needs of the community, on the trend of legislation, and to a certain extent on the working of the lay mind; they can also foresee the possible results of a course of action, because they are composed of men who have years of experience to guide them.

The plea is one for understanding by Branch councils one of another, and for trust in Branch councils by members and groups of members of the Branches. The Branch councils (and the central body, the Federal Council) can be trusted to realize that the medical profession must adjust its attitude to the changing economic and social conditions of the present day. They know that they will be most effective if they foresee changes and initiate them before they are made obligatory.

Current Comment.

DISEASE IN ETHIOPIA.

WHEN Mussolini's armies invaded Ethiopia it was generally considered that the danger of death and injury from Haile Selassie's antiquated weapons would be among the least they would have to face. Ethiopia was said to be a plague spot. Speculations were made that those Italian soldiers who did not succumb to the onslaught of Ethiopian arrows and other missiles would fall victim to disease. Some of this was true enough. There was not much public health work in Ethiopia before the Italian invasion. Authentic statistics of the prevalence of various diseases in that country are therefore of interest. Some figures have recently been published by J. W. S. Macfie, who was attached to a British Red Cross unit at Waldia, where there had previously been no medical service.[1] Waldia is in mountainous country and is 5,000 to 6,000 feet above sea-level; it is damp and, at night, cold. Macfie states that his unit went to Waldia primarily to attend to those who had been wounded during the bombardment of the town by Italian aeroplanes. Apparently the populace considered this too rare an opportunity to miss, and large numbers asked for treatment. During twelve days, from January 21 to February 1, 1936, 989 patients attended. Of these, 209 are set down in Macfie's list as suffering from "minor injuries, abscesses, ulcers, boils, septic sores, etc." The more interesting figures (quoted from Macfie's list) are as follows:

Tape-worm infestation	133
Rheumatism, myalgia, lumbago, etc.	100
Syphilis	89
Dyspepsia, gastric catarrh	52
Eye conditions (mostly conjunctivitis)	50
Scabies	47
"Mossy" foot	29
Gonorrhœa	25
Impetigo	24
Malaria	20
Teeth, carious	20
Diarrhœa	17
Injuries by bomb, gun or sword	16
Ear conditions, mostly otitis	15
Tuberculosis: lung, glands, etc.	8
Anæmia, debility, etc.	7
Dysentery (amœbic)	7
Leprosy	4
Elephantiasis: leg, scrotum	4
Hernia: inguinal	2
Prostatic enlargement	2
Mollusca fibrosum	2
Goitre, . . . Appendix abscess, . . . Epilepsy, . . . Morbus cordis, . . . Paralysis agitans, . . . Goundou, of each	1

Macfie states that in many cases the disease was very advanced, producing appearances that are never seen nowadays in any civilized country. The tertiary syphilides were perhaps the most remarkable. "Some of the prodigious gummata were indeed impressive, and satisfactory, in that they responded to treatment very readily." At Dessye, some 75 miles from Waldia, there is a medical mission, which has been active for many years. Doubtless some news of its work had reached the mountains of Waldia; for many of the people there demanded injections, insisting that they had syphilis, "often", says Macfie, "quite untruly". This simple faith in injections is not confined to Ethiopians. In Melanesian countries, for example, where arsphenamine is used with dramatic success in the treatment of framboesia, natives ask for injections for all sorts of complaints; even a prick with a needle to obtain a blood film may make them feel that they have had some highly efficient treatment. Among more enlightened people, the demand for something to be injected is one of the reasons for the innumerable beautiful ampoules that have such a vogue today.

Only 133 patients were treated for tape-worm infestation. There was insufficient of the appropriate remedies on hand for all sufferers to be attended. When fresh stocks were obtained, some 30 patients were treated each day. Macfie states that "officers of the Imperial Guard soon showed an inclination to send us whole companies of their men for treatment, and our resources would not stand that!" As raw meat is a favourite article of diet, tape-worm infestation is "almost an institution". The drinking of *koso*, the inhabitants' anthelmintic, is "a periodical social, if not also religious, rite". It would seem that the Ethiopian who does

[1] *Annals of Tropical Medicine and Parasitology*, October 21, 1936.

not possess a tape-worm can scarcely be regarded as a normal Ethiopian.

There are apparently several diseases known as "mossy foot". The type seen at Waldia was similar to that called by Loewenthal "lymphostatic verrucosis"—a self-explanatory term. The ætiology is obscure; a number of different causes are possible. Macfie notes the rarity of hernia at Waldia as compared with West Africa. He suggests that this may be accounted for by the fact that Ethiopians at Waldia do not carry heavy loads on their heads from childhood, as the West Africans do. Typhus and relapsing fever do not appear on the list; but Macfie states that they were probably common. It is possible that there were many patients with other serious diseases in the district; it is unlikely that many people who were seriously ill would be able to attend as out-patients, especially in a country with primitive means of transport.

It is evident from Macfie's report that the prevalence of metazoal, protozoal and bacterial diseases in parts of Ethiopia was high before the Italian invasion. It would seem that the Italian health authorities have a heavy task before them. If Macfie's experience is an indication, they should at least have little trouble in persuading patients to undergo treatment; hygienic reforms are another matter.

ANÆMIA AND DIGESTIVE FUNCTION.

"NOR is it difficult to see how fatal anæmia must follow an amount of degenerative disease reducing the amount of gastric juice so far that the assimilation of food is rendered wholly inadequate to the wants of the body." This quotation betrays its age in its language, but nevertheless the general proposition that it states has been proved true of recent years to an extent that would have surprised Austin Flint when he made this prophetic statement in 1860. George R. Minot, in surveying the relationship between anæmia and the gastro-intestinal tract, points out that an association of anæmia with the processes of digestion and absorption has interested medical investigators for over one hundred years.[1] He further quotes C. H. Jones, who made histological studies of the stomach fifty years ago and who recognized atrophic changes as possible results of depraved nutrition. In reviewing the now familiar mass of facts concerning deficiency anæmias, Minot examines the claim of Greenspon that the gastric factor that is deficient in macrocytic anæmia is a hormone which acts directly on blood production, but he considers this view to be ill-founded for various experimental reasons. One point which he mentions is of particular importance. In most cases of macrocytic anæmia residual amounts of the intrinsic factor are present which are able to maintain a supply of blood cells at a lower level than the normal. It is even possible for a higher blood level to be maintained simply by utilizing a slight increase in the intrinsic factor, which may become available by the use of a thoroughly liberal diet, although, of course, as is well known, a normal blood state cannot be restored or maintained without additional amounts of the intrinsic factor. This fact is a simple illustration of the necessity for an ample diet. The same applies with equal force to anæmia of the hypochromic type, in which a poor diet, infections and anomalies of digestion and absorption all contribute to the vicious circle of hæmoglobin deficiency. As Minot states, it is common to find in the so-called "idiopathic" hypochromic anæmia of women that the patients have had gastro-intestinal symptoms for years, have been more or less invalids and have partaken of very inadequate diets for a long time. In connexion with changes occurring in the gastro-intestinal tract either as the cause or as the result of severe anæmia, a clear distinction must be drawn between those which are nutritional and remediable and those which are of chronic inflammatory nature and not always capable of complete cure. In this connexion it is curious how incomplete our knowledge really is, for in the so-called Plummer-Vinson syndrome, in which there are changes in the œsophagus with difficulty in swallowing, there are undoubted changes in the epithelial and sub-epithelial structures, yet full doses of iron cause almost complete restoration to health, whereas achlorhydria is usually persistent. In truth, as Minot says, the factor responsible for the gastro-intestinal lesion is not exactly known. Other interesting changes which may be actually seen to disappear under our very eyes are the atrophic lesions in the skin and nails, since these are easily cured by the administration of iron. Though it is possible only to refer in a brief and disconnected way to the very many interesting points raised in this paper, one other which may be mentioned is the need for some method by which defective absorption from the digestive tract can be measured. This may affect the utilization of substances of both organic and inorganic composition, but the author points out that delays in iodine absorption may readily be measured, and that these form some rough indication of the degree of disturbance of the absorptive capacity of the small intestine. All the iron present in food is, of course, not fully available for absorption, and less than 50% is sometimes actually utilized. Factors which favour this absorption are the presence of readily oxidizable substances and an acid reaction; on the contrary, certain mixtures of food may act so as to render the iron insoluble. Chlorophyll appears to have some synergic action with iron, but this is one of the many details of iron metabolism which have not yet been fully worked out. However, there remains still an important principle, namely, that deficiency anæmias do not arise if the digestive tract is healthy, if the diet is correct for the period of life, and if other disturbing factors, such as blood loss or infection, are absent.

[1] *American Journal of Digestive Diseases and Nutrition*, November, 1936.

Abstracts from Current Medical Literature.

RADIOLOGY.

Ureterocele.

E. R. WILLIAMS (*British Journal of Radiology*, January, 1936) feels that, if radiologists were familiar with the anatomical pathology of the condition, radiological study would in many more cases indicate the presence of ureterocele during the course of intravenous urography. In approximately 50% of ureteroceles one or more calculi are present in the cyst. Calculus formation follows cyst formation rather than contributes to it. Given the presence of a calculus within the ureterocele, the following points should enable the radiologist to suspect the anatomico-pathological condition present. (i) The presence of a calculus in close relation to the terminal portion of the ureter, which probably shows some degree of dilatation. With an opaque medium in the bladder the calculus should be projected within the bladder shadow, whatever technical angulation is used. The calculus is relatively fixed in position, whatever posture is adopted by the patient. (ii) Presence of a halo surrounding the calculus, caused by the cyst wall, with soft but distinct margins. If the ureterocele does not contain a calculus, the following points should be considered: (i) Presence of a sausage-shaped or spherical filling defect near the trigonal region, approximately in line with the terminal extravesical ureter. (ii) Presence of a fine halo about the filling defect, sufficient to cast a sharp intervening contrast line between the opaque urine in the bladder and the opaque urine in the ureterocele. (iii) The ureterocele may tend to fill fully before a dense vesical shadow is noted, and also it may retain opaque urine when the bladder has been emptied. Common to both circumstances the following associations might be expected: (i) variable degree of ureteric dilatation, (ii) possibly hydronephrosis, (iii) upper urinary tract calculus formation, (iv) presence of anatomical variations in the ureter and renal pelvis. Attention should be paid especially to the appearances of the terminal portion of the ureter and bladder in the fifteen to thirty minute period following intravenous injection. After bladder filling is well established, it is possible that the vesical opacity would be too great to enable appreciation of the lesser degrees of the condition. Serial studies could then be made with different degrees of micturition, and suspicious findings could be confirmed by an aero-cystogram.

Asbestosis.

J. RUSH SHULL (*Radiology*, September, 1936) states that the Röntgenographic picture of silicosis does not resemble asbestosis. In the former the upper third of the lungs is involved and the fibrosis is parenchymal and not interstitial. The lung fields in silicosis show characteristic nodulation, and in advanced cases a coalescence producing dense opaque areas in contrast with the typical, hazy, ground-glass appearance of the lung fields in asbestosis. Silicosis is a progressive disease, even when the patient has been removed from dust exposure. This is not the case in asbestosis. Also in asbestosis there is a tendency to pleural and pericardial involvement which is strikingly absent in silicosis. Asbestosis does not predispose to tuberculosis.

The Radiological Appearance in Ureteritis Cystica.

FOLKE KNUTSSON (*Acta Radiologica*, Volume XVI, Part I, 1935) describes two cases showing changes in the appearance of the ureters typical of *ureteritis cystica*. In this condition multiple epithelial cysts are present in the ureteral wall. Similar formations also occur in the renal pelvis (*pyelitis cystica*) and the urinary bladder (*cystitis cystica*); these lesions occur most commonly in the last-mentioned site. The cysts may be located at different depths in the wall. When reaching a certain size they bulge into the lumen. If the cysts are scanty and of small size they are probably of no clinical significance. If present in any greater number, or if of greater size and located at narrow parts of the efferent urinary tracts, then they may conceivably give rise to obstruction. The cysts are best seen after retrograde pyelography, but may also be seen after intravenous urography. They show as closely-packed, small, round, filling defects due to a nodular relief of the mucous membrane. The number and size of the cysts may vary considerably. In some cases the whole wall is closely studded with nodules, while at other times it may be the seat of a few sparse cysts. If they occur very sporadically or are solitary, it is more difficult to make a diagnosis. In the differential diagnosis one has then to consider urate concretions, papilloma or polypus. One must also guard oneself against confusion with air bubbles introduced into the ureter at the time of injection.

Pulmonary Silicosis in Porcelain Workers.

GUNNAR JONSSON (*Acta Radiologica*, Volume XVI, Part IV, 1935) places great value in the lateral view of the chest in silicosis, particularly when it is necessary to differentiate the condition from tuberculosis. The most severe silicotic changes occur in the area of distribution of two bronchial branches, namely, the dorso-lateral branch of the bronchus to the upper lobe and the first dorsal branch of the bronchus to the lower lobe. If tuberculous processes are also present, the picture is altered to a greater or less degree, according to whether the lesions of silicosis or tuberculosis predominate. The changes due to fibrotic tuberculosis seldom give a similar picture. The accumulation of dust in certain parts of the lung can be explained in two ways. It may be that more dust is inspired into those areas, owing to varying pressure conditions in different parts of the lung; or conceivably the dust is at first uniformly distributed in the lung and afterwards removed from certain parts by coughing *et cetera*, while in other areas the particles are retained.

Otto's Disease and Other Forms of Protrusio Acetabuli.

KRISTIAN OVERGAARD (*Acta Radiologica*, Volume XVI, Part III, 1935) states that under the designation of "*protrusio acetabuli*" a number of cases of deformities of the pelvis are described, the chief feature of each being that the part of the pelvis corresponding to the inside of the acetabulum portrudes more or less deeply into the lumen of the small pelvis, deforming and narrowing it. Besides the floor of the hip socket being pushed further in towards the median line of the pelvis than is normally the case, the contents of the hip socket (that is, the head of the femur) are also embedded more medially than they normally are, and a part of the neck of the femur is lodged in the deepened hip socket. In the literature the designation *protrusio acetabuli* is applied to diseases very different in nature. The conditions so designated may be divided naturally into three groups: (i) Secondary protrusions: The condition arises as a complication following upon a clearly defined focal disease in the hip joint, or upon a trauma. (ii) Otto's disease or osteoarthritic protrusion: The disease is regarded as a special form of the deforming osteoarthritis in the hip joint. Most often the disease appears in both sides. A certain form of development of the hip socket (deep hip sockets) is thought to be a contributing factor, or the chief cause of the development of the disease. (iii) Juvenile osteoasthenic protrusion: The disease is developed at the age of puberty in women, with no signs of arthritic or traumatic changes in the hip joint, probably as a result of weakness of the bone tissue.

PHYSICAL THERAPY.

Radiation in the Treatment of Cerebellar Medulloblastomata.

ELLIOTT C. CUTLER, MERRILL C. SOSMAN AND WALTER W. VAUGHAN (*American Journal of Roentgenology and Radium Therapy*, April, 1936), comparing the cystic astrocytomata of the cerebellum and the cerebellar medulloblastomata, found that both occurred most frequently in children, often giving identical symptoms and signs; the prodromal period in the astrocytoma, however, may be considerably longer than in the medullo-

blastoma. The cerebellar medulloblastomata are in the great majority of cases situated in the mid-line. The few cases found in adults are more likely to be laterally situated in the cerebellar hemispheres. The most valid objection to radiotherapy without operation, and a logical one, is that if the patient survives it can never be proved that he actually harboured the tumour in question. However, the authors accept the epithet of "unscientific" or "unconfirmed" work and do it willingly. The importance of irradiating the spine and ventricular system is verified clinically by the classification of cases showing the prolongation of the average duration of life with the development of present technique. In conclusion, after examining eighty-one cases, the authors state that X ray therapy offers the only hope for the cure of medulloblastomata. It may be given without a decompression. Irradiation may be used in certain selected cases as a therapeutic test for differential diagnosis in medulloblastoma suspects. X ray therapy to the ventricular system and the entire spine as well as to the cerebellum is essential.

X Ray Treatment of Gas Gangrene.

JAMES F. KELLY (*Radiology*, January, 1936) recommends that X ray therapy be given in all cases of gas gangrene. The dose required is so small that almost any X ray machine is capable of giving the treatment. Treatments should be started no matter how hopeless the case may seem. They should be given morning and evening or twice a day for three days, and should be of sufficient voltage to insure penetration of the involved tissue—about 90 to 100 kilovolts on the extremities, from 130 to 160 kilovolts on the trunk, and 100 *r* units per dose. It would appear that amputation is detrimental to the welfare of a patient with gas gangrene of an extremity and that it is safe to rely entirely on serum, local antiseptics, local minor surgery, and X ray treatment. Amputation should be undertaken only when the tissues are hopelessly damaged by causes other than gas gangrene. Finally, the procedure is based entirely upon clinical evidence, and more work must be done before the exact status of X ray therapy in this condition is determined. However, some work done at Stanford University on radiation reaction in living tissues leads to the conclusions that, following radiation, hydrogen peroxide is formed in the tissue radiated. If this is true, and since, as we all know, these organisms are anaerobic, this formation of hydrogen peroxide may be the answer to the problem.

Fever Therapy.

ARTHUR U. DESJARDINS (*Archives of Physical Therapy, X-Ray and Radium*, April, 1936) mentions, among the various physical methods of fever therapy that have been tried, hot baths, conventional diathermy, short wave diathermy, radiothermy, radiant light (infra-red generator and carbon filament lamp), inductothermy and air-conditioned chambers (Simpson Kettering). Hot baths can be used effectively when the period of fever need not be long, but when it is desirable to maintain the fever for an extended period this method becomes distressing and debilitating for the patient. Diathermy has been employed effectively, but requires the application to the patient of large ventral and dorsal electrodes, which must be carefully maintained in contact with the skin over a large area. Short wave diathermy has been used with considerable success. Until simpler and more effective means of inducing fever were available, long and short wave diathermy were the best methods. The use of short wave apparatus, however, has disadvantages that have caused many to give it up. Not only is the apparatus expensive and somewhat complicated, but the tendency of the current to arc towards pools of perspiration and thus to cause burns makes it necessary to provide accessory equipment to evaporate the sweat as rapidly as it emerges from the skin. Radiant light fever chambers are being used with a certain amount of satisfaction. They are less expensive than certain other forms of apparatus designed for this purpose. Of the different forms of physical apparatus thus far designed for fever therapy, the one which seems the most effective is the air-conditioned chamber known as "Kettering hypertherm". With it the temperature can be raised more rapidly than with radiant light chambers, and any degree of fever can be attained and maintained for as long as the condition of the patient allows or requires. The patient's body (except the head, which is outside) is entirely free within the chamber, and the apparatus does not involve the use of contact electrodes, condenser plates or other electrical appliances. Fever therapy is a fairly strenuous test of cardio-vascular function; therefore, patients over sixty years of age, or of any age who may be suffering from cardiac or renal disturbances, should not be subjected to it. The author discusses other conditions in relation to fever therapy.

Malignancy in the Skin.

J. F. BROMLEY (*The British Journal of Radiology*, October, 1936) confines his remarks to the treatment of epidermoid cancer, sarcoma and melanotic sarcoma. The three main histological varieties of skin cancer are discussed: squamous-celled epithelioma or acanthoma, basal-celled epithelioma, and transitional or malignant lympho-epithelioma of Regaud. It is emphasized that any proposed method of treatment of skin cancer must include, first, elimination of any source of chronic irritation and, secondly, special attention to the growing edges of the tumour; thirdly, the treatment must be adequate, so that the growth will not reappear and become radio-resistant after inadequate or wrongly spaced dosage; fourthly, some method must be found to deal adequately with the lymphatic glands. The writer considers it a mistake to advocate one method of treatment as applicable to all malignant tumours; every tumour should be considered as an individual problem. For interstitial radium treatment he calculates his dosage in Röntgen units, and he states that rodent ulcers usually require 3,000 to 4,000 *r* for complete healing, while squamous-celled epitheliomata require at least 6,000 *r*. An account of twelve months' experience with the Chaoul contact therapy X ray tube is also given. The author's doses with this type of therapy are 400 *r* daily up to 2,000 *r* or more for rodent ulcers, and 6,000 to 10,000 *r* for squamous-celled carcinomata. Treatment of carcinoma of the vulva and penis by this method is very satisfactory. Disadvantages are that small fields only can be used in contact work (the intensity falls off too rapidly at the edge of large fields) and that it is essentially a surface method and not to be advocated for lesions going deeper than 2·5 centimetres. Treatment of epithelioma supervening on radiation dermatitis is surgical, and any further radiation, even ultra-violet radiation, is contra-indicated. In melanotic sarcoma endothermy is the treatment of choice, with X ray and radium therapy as adjuvants. For scrotal carcinoma (common in Manchester, where the lecture was given) surgery is the method of choice, with, possibly, radiation treatment to the inguinal glands.

The Rôle of Physical Therapy in Fractures.

O. B. BOLIBAUGH (*Archives of Physical Therapy, X-Ray and Radium*, October, 1936) discusses the development of physical therapy, emphasizing how much we owe to the work of Championnière and to lessons learnt in the Great War. Fractures do not heal with any degree of uniformity. The formation of callus is influenced by the age of the patient, the location and type of fracture, local conditions about the fracture and the type of treatment. Union occurs most rapidly in cancellous bone. These things must be taken into account in prescribing physical therapy. Only the simple types of physical therapy, that is, massage, heat and exercise, are needed. Light massage is used for its soothing and relaxing effect, preliminary to the movements of joints; deeper massage to aid the lymphatic and venous systems in carrying off the products of œdema and hæmorrhage. Heat is soothing, relieves pain and also causes increased flow of blood to the part. Movement may be active, passive, assistive, resistive. Therapy in a few of the more important fractures is discussed. The importance is emphasized of always immobilizing joints in a position of maximum function and of using gravity as an aid to the recovery of motion and not as an impediment.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at Hamilton on November 21, 1936, DR. WALTER SUMMONS, the President, in the chair.

Multiple Arthritis.

DR. P. B. HOUGHTON showed a male patient, a boot repairer, thirty-two years of age, with swelling and stiffness of all the small joints of the hands and the feet, wrists and ankles, shoulders, hips and spine, and to a less extent the knees. Two years before the meeting he had developed painless swelling in both feet in and around the ankle joints, more pronounced when he had been standing all day. Six months later he had developed pain and stiffness in the wrists with swelling of the joints; the shoulders also became involved, then the hands, knees and later the hips and spine. Soon after the onset of pain in the joints the patient developed pyrexia with a temperature up to 102° F., a pulse rate of 120, shivers and night sweats. Physical examination failed to reveal the cause, but on examination of the urine it was found to be loaded with pus cells and motile bacilli, although the patient had had no pain on micturition and only very mild frequency. The pyelitis cleared up under treatment and the patient lost the joint pains and resumed work in October, 1935, apparently quite well.

In February, 1936, he had a recurrence of the rheumatic manifestations and at times ran a mild temperature up to about 99·6° F. Examination of the urine on three occasions failed to reveal pus cells or motile bacilli. On March 16, 1936, the patient complained of severe pain in the left side of the chest, round the region of the apex of the heart; the pain was worse on coughing or deep breathing. He had a temperature of 102° F. and a pulse rate of 120; he looked very ill and was having sweats and rigors. On examination there was a friction rub over the site of the pain, synchronizing with the heart beat and accentuated on inspiration. The heart dulness extended out on the left side to four and a half inches from the mid-line, with the apex beat in the sixth intercostal space. The heart sounds appeared to be clear. The patient developed dulness at the left base, and on exploration about half a pint of straw-coloured fluid was withdrawn. The pleurisy gradually subsided but during the subsidence he developed a systolic bruit at the mitral area. Skiagrams that were taken at that time showed an enlarged heart shadow with dulness of the lower portion of the left lung.

Dr. Houghton said that towards the end of the patient's stay in hospital, about four weeks after the onset of the condition, he had developed a painless, symptomless jaundice, which lasted for about three weeks. An attempt at cholecystography when this subsided was unsuccessful, as the gall-bladder did not show up at all. A blood examination was made during the attack; the red cells numbered 4,200,000 and the white cells 12,500 per cubic millimetre; the hæmoglobin value was 70%; the film was normal. During the patient's previous attack skiagrams of his kidneys showed a dense shadow at the upper pole of the left kidney, and pyelography following the attack revealed no abnormality in the pelvis or the ureters.

Dr. Houghton went on to say that since his last acute illness the patient's rheumatoid condition had become worse. Examination of the patient's urine during the two months before the meeting had revealed the presence of pus cells and motile bacilli. Dr. Houghton said that a skiagram of the paranasal sinuses had shown fluid in the left maxillary antrum; the antrum was washed out, but only thin fluid was obtained and no pus. The patient had had no symptoms of antrum infection except a cold in the head six weeks previously; five and a half years before he had had his tonsils removed by dissection, and five years previously Dr. Houghton had carried out cystoscopy because of a suspected stone. A stone protruded from the opening of the right ureter; Dr. Houghton had been able to dislodge it and the patient had passed it later in the day.

Dr. Houghton said that the patient was being treated with a course of "Solganol B. Oleosum", but had up to the time of the meeting shown little improvement. The cholecystogram had been repeated with more success, but no abnormality was revealed. At the time of the meeting the red blood cells numbered 4,500,000 and the white cells 8,000 per cubic millimetre; the hæmoglobin value was 70%; the film was normal.

DR. G. A. PENINGTON advocated taking a wide view of the general condition of Dr. Houghton's patient; attention should not be concentrated on the treatment of the septic foci. It was generally admitted that septic foci played some part in the condition, but whether they were instrumental in causing the joint damage was controversial. Removal of septic foci was sometimes followed by improvement, but in many instances the expected improvement was not soon apparent. He thought that the general state of the man's nutrition would have to be improved before removal of septic foci alone would result in healing of the joint condition. Urinary infection was definitely proven and should be dealt with conservatively. Dr. Penington admitted scepticism as to the value of urinary antiseptics, which in his hands had been practically valueless. The alteration of the hydrogen ion concentration of the urine to render the urine bacteriostatic by the use of mandelate and ammonium chloride was worthy of consideration, and sometimes nitromuriatic or hydrochloric acid helped to overcome urinary infection. He was of opinion that the administration of some hydrochloric acid might help the patient, as in such cases lowered acidity was nearly always present. He did not think that treatment of the antrum would make much difference. The patient was still a very sick man and required a high caloric diet with a large amount of fluid; if necessary, some insulin could be used to help in the utilization of carbohydrates. A high vitamin content was essential, and as there was some evidence of decalcification, the use of vitamin *D* with calcium should be of some value. Dr. Penington summarized his advice by saying that the patient must remain in bed and be given a high calorie diet with ample fluid; when the general condition was attended to first in this way local measures could be adopted later to deal with specific symptoms.

DR. T. G. MILLAR thought that the skiagrams of the antra indicated grossly swollen mucous membrane, and he could not agree that a fluid level was present. He would suggest that the antrum should be washed out four or five times, and if no macroscopic pus was present, no more need be done. The problem of rhinology today was to know what to do with an antrum showing radiological dulness but no definite infection. The opinion he had expressed would be given by 80% of rhinologists, but the other 20% would advocate radical antral surgery.

DR. J. G. R. FELSTEAD said that the medical profession would be disappointed if it gave up entirely the conception of the infective nature of arthritis, though the colon bacillus was not commonly associated with joint trouble. The colon bacilli in the urine might be covering an infection with staphylococci or with streptococci, which might be overlooked. If they were going to hold on at all to the infective theory, they had in this case a focus of infection in the kidney, and behind it all there was a stone. There was often an infective origin for calculus formation, and the infection was often perpetuated by the stone. The sooner the stone was removed, the sooner the infection might be cleared up, and he advised that the stone should be removed when the general condition of the patient permitted the operation.

Dr. Penington at this stage said that he had been interested in an article on acid-base diet in renal calculosis, which he thought had appeared in the Cleveland number of *The Surgical Clinics of North America*. It was claimed that as a result of the acid-base diet calculi which had been shown to be present radiologically were not evident in subsequent films. Dr. Penington admitted that it was difficult to accept this claim. He also mentioned that extreme care was necessary in the administration of heavy metals, such as gold; if gold was to be used for this patient, the dose should be small and the interval

should be prolonged. High carbohydrate diet with insulin should be given on the day of injection, and five to ten cubic centimetres of defatted milk could be given intramuscularly to counteract non-specific shock.

DR. KEITH ROSS said that one kidney was still normal and every effort should be made to keep it normal; if treatment was delayed till the second kidney was infected, the delay would be too long. He quoted a number of authorities who were opposed to the conservative treatment of an infected kidney. He suggested that possibly sympathectomy might be of value, and he would like to hear the opinion of Dr. Coates on this aspect of the subject.

DR. A. E. COATES stated, in reply to Dr. Ross, that he considered the chronic arthritis to be of infective origin; and as there was a possible cause in the kidney, the calculus should be dealt with. The arthritis would run its course, and during the acute stage the wrists should be splinted. Sympathectomy had been performed for this condition by other surgeons, for example at the Mayo Clinic and recently by Young, of Glasgow, but time alone would indicate whether the results were permanently beneficial. During a recent visit to the Mayo Clinic, Dr. Coates had observed that there appeared to be less enthusiasm for the operation than he would have expected. It was to be noted that removal of the stellate ganglion deprived the upper limb of vaso-constrictors, but Lewis had shown that it was not quite so simple, for probably the vaso-dilator mechanism was also involved. The limb became warmer in cold weather, but the normal response to external cold was no longer present. Whether it was wise to deprive a limb of its normal responsive mechanism to external temperature in the absence of such a gross condition as the destructive lesions of severe Raynaud's disease was debatable. Dr. Coates thought that due regard should be paid to all aspects of the disease, and operations which were of doubtful benefit should be undertaken with caution.

DR. DOUGLAS GALBRAITH described the end result in a somewhat similar case, that of a girl, fifteen years of age. Five years earlier she had had middle ear disease with lateral sinus involvement and mastoid trouble. The blood stream had been infected and she had developed multiple infective arthritis, which had gone on to osteoarthritis. The girl had been admitted a year before the meeting to the Frankston Orthopædic Branch of the Children's Hospital with complete fixation of the ankles, knees and many joints of the hands. The infection cleared itself out, but the end result was deplorable in spite of the inactivity of the infection. Dr. Galbraith agreed with Dr. Penington that the general condition should be improved and then the kidney infection might be cleared up.

Dr. Houghton, in reply, said that the patient had been on a highly nutritious diet, but had not had any hydrochloric acid. He had had a series of six milk injections, varying from one cubic centimetre to ten cubic centimetres, and had had a severe reaction from the last injection. He had not had more than 0·01 gramme of "Solganol" at any time. The discussion had been of value and he felt now that if the patient's general condition could be built up, an attempt might be made to get rid of the stone and there might be hope for some improvement in the joint condition.

Chronic Colitis.

DR. J. LE M. KNEEBONE showed a male patient, forty-six years of age. When first seen eight years previous to the meeting he had complained of not being well since an operation which had been performed for appendicitis nine years before; he said that he could not stand much exertion and that he got attacks of pain on the right side of the abdomen, below the navel, and that sometimes the pain extended to the left side. He said that immediate relief from the pain resulted if his bowels opened; at the end of a motion blood and slime would be passed; if the pain had been severe, a larger amount of blood was passed. The patient said that the pain did not last for more than fifteen minutes and would not return for several days; attacks were more frequent if the patient was at all constipated. There had been no vomiting and he had a good appetite and slept well. Physical examination had revealed no abnormality; he had been given liquid paraffin and was asked to report for further examination. He had not attended the hospital again for eight years, five months before the meeting, when he had complained of a severe pain after defæcation. He reported that he had had comparatively little trouble with his bowels in the intervening eight years.

Dr. Kneebone said that the pain was apparently due to an anal fissure which had been dealt with by operation. Soon after the operation the patient had had an attack of abdominal pain, frequent loose motions containing bright red blood, and a temperature which rose to 102° F. He continued to run a temperature from 101° to 104° F. for one month with two to six bowel actions a day; the motions had been brown fluid, offensive and contained bright red blood. No abnormality was detected in the rectum, but one superficial ulcer covered with a grey slough was found in the lower sigmoid. Neither emetine nor antidysenteric serum produced any benefit, examination of the stools failed to reveal tubercle bacilli, and the Widal test also gave no reaction. Dr. Kneebone said that no further bacteriological investigation had been made for three weeks after the onset of the acute symptoms. Bargen's vaccine (100,000,000 organisms per cubic centimetre) had been administered every three days in a gradually increasing dose. Whether as a result of the vaccine or not, the temperature began to subside and became normal a week later. Starch and opium enemata (four ounces) were given at the beginning so that the patient might obtain rest at night, and later simple saline wash-outs were used every day. A barium enema was given a week after the temperature had reached normal and the motions were reduced to two a day. The films showed changes limited to the caecum and the descending colon, especially the latter, in which extensive changes had occurred.

The patient was discharged from hospital on August 17, 1936, two months after admission, on a diet consisting of frequent small meals free from meat and cellulose. Dr. Kneebone said that the patient, although able to get about, had not felt strong and had had occasional attacks of abdominal pain with bowel actions, when the stools contained blood. He would like to hear a discussion on two particular aspects of the case, namely, the pathological significance of the radiological appearances and, as the patient at forty-six years of age appeared to be condemned to semi-invalidism, the justification for radical operative interference.

DR. ARTHUR MORTON considered that examination of the skiagrams shown at the meeting indicated that the colon was in an abnormally high position and that there was some stenosis of the descending colon; a number of small diverticuli could be seen in the transverse colon, and irregular edges appeared in the lower portion of the descending colon; he thought that several round polypoid growths or fibromata must be present. As the patient had had melena, presumably the bleeding had come from diverticulitis or from ulcers, but the skiagrams did not show the picture typical of ulcerative colitis, in which, when the colon was distended, it should look straight, with an absence of haustration.

DR. G. A. PENINGTON said that the patient produced a symptom complex in which he had been interested for many years. It was surprising that two such vexed problems as that of chronic arthritis and of chronic bowel lesions should be under discussion that afternoon. In the case of the patient shown by Dr. Kneebone, Dr. Penington did not think that a diagnosis of diverticulitis was sufficient. Although there was radiological evidence of the presence of diverticuli, the patient's history and the findings in the descending colon and the bowel symptoms could be dated back to the appendicectomy some seventeen years earlier. It was notorious that bowel inefctions were extremely variable, with periods of quiescence, but in the present case there were lesions which could be seen and which had been seen by sigmoidoscopy; it could be assumed that there were ulcers elsewhere. Dr. Penington thought that the type of infection which had caused the ulceration was a debatable matter; it was necessary to

exclude amœbic infection, if possible, although the patient had never been outside Victoria; Dr. Penington and his brother, whose work brought him in contact with tropical medicine, had had such cases and had found the protozoa by repeated examination of scrapings from the side of the ulcer. Lack of response to a course of emetine was not sufficient to exclude amœbiasis; this form of treatment should be continued for at least three months. The patient was definitely more sensitive to thumping over the liver than he was to similar examination on the left side. If no amœbæ could be found, a pathogenic organism capable of causing the condition would be more likely to be isolated by pathogen-selective culture than by any other method. In this way a hæmolytic streptococcus might be obtained, and even if the condition was proved to be amœbic, the attempt should be made to isolate streptococci. Anti-amœbic measures alone would not do. Specific treatment with an autogenous streptococcal vaccine should be included if possible, and the patient should be placed on a high caloric diet rich in vitamins to revitalize the mucous membrane. These patients needed protein, and undue restriction of meat was harmful; the patient's resistance was lowered thereby. Milk should be restricted, and if its use was not harmful, small amounts, predigested and cooked, might be given. Raw milk was one of the least well tolerated foods in these cases. The patient had had diarrhœa as recently as two weeks before coming into hospital, so rest in bed was indicated until the symptoms were alleviated. Dr. Penington expressed sympathy with Dr. Kneebone for having to treat such a trying condition, but he besought Dr. Kneebone to go on along the lines indicated; Dr. Penington was definitely opposed to surgical measures.

DR. A. E. COATES considered that the probable ætiology of the condition and the line of treatment suggested by Dr. Penington were correct; he had nothing much to add except that he too considered surgery inadvisable for the chronic colitis. The radiography certainly suggested the condition of chronic infection, especially in the left colon. There was the history of pain following appendicectomy and some distension of the abdomen. The coils of the small bowel appeared to be dilated and he was not quite sure that a mild chronic obstruction of the small intestine could be excluded. While he agreed that the condition in the colon did not require any surgical treatment, as the patient appeared to be doing well, Dr. Coates was not able to dismiss the possibility of a second lesion in the abdomen, and since the mortality of acute small bowel obstruction was high, the patient should be observed in case further symptoms should call for laparotomy.

Dr. Kneebone, in reply, expressed his thanks to those who had contributed to the discussion. He felt that if the condition was due to streptococci or amœbæ, with the production of such extensive changes, the patient would be in trouble for the rest of his life. It might be possible after a reasonable time to deal with a limited portion of the bowel. He had not had meat or milk, but had been getting a generous supply of vitamins. Dr. Kneebone would restore meat to the diet and attempt the bacteriological investigation, though he had to admit that there were practical difficulties in a country centre such as Hamilton.

Hydronephrosis and Renal Sympatheticotonia.

DR. S. C. FITZPATRICK showed a female patient, forty-six years of age, with left-sided hydronephrosis which was possibly due to renal sympatheticotonia. For twelve months she had suffered from attacks of left-sided renal colic. At first the attacks were at an interval of eight weeks, but for two months the interval had been reduced to three weeks. The pain began in the left costo-vertebral angle and spread down to the left groin, growing steadily worse. She said that "A.P.C." tablets would sometimes stop the pain. The pain usually came on two days before the menses; the patient had also complained of severe attacks of "hay fever" for the past eight years, beginning each year in November. Her first attack for 1936 had begun a week before the meeting and had lasted for one day; flatulence, constipation and frequency of micturition had accompanied the attack. Dr. Fitzpatrick said that the patient had complained of nasal obstruction, persistently dry, scaly and cracking skin on the face, forearms, hands, knees and on the soles of the feet; also piles and vaginal birth injuries. The patient's menses had been regular, the interval being twenty-two days; the last period had extended for six days and was profuse. The patient had six children living and two dead; one had died of "cancer" at three years of age, and one had died of "convulsions"; the youngest child was four years of age. She had had one miscarriage seventeen years before and an abortion three years previous to the meeting. On examination the patient had facial asymmetry, the left side being smaller than the right. The temperature was 98° F., the respiration rate 14, and the pulse rate 84; the patient also had soft arteries. In addition, she had nasal obstruction due to septal deviation, and was breathing through her mouth; she had a tendency to drooping eyelids, and the skin was ichthyotic. Nothing abnormal had been detected in the heart, lungs or abdomen; the systolic blood pressure was 108 and the diastolic pressure was from 66 to 42 millimetres of mercury. Nothing abnormal had been detected at the vaginal examination, and the Wassermann test gave no reaction.

Dr. Fitzpatrick said that during the attacks there had been tenderness in the left costo-vertebral angle; the urinary findings had been normal, except on one occasion, when blood cells were found on microscopic examination. A hypodermic injection of one one-hundredth of a grain of eserine salicylate had completely relieved the attack. Skiagraphic examination had revealed no signs of calculi in the urinary tract, and cystoscopy on February 25, 1936, revealed a normal right ureteric orifice and a smaller rounded left ureteric orifice. Pyelography revealed a left hydonephrosis of the pelvic type. As it was considered that the left ureteric orifice was smaller than normal, it had been dilated and a catheter was left in for three days and a colloidal silver solution was instilled periodically. The patient experienced a severe attack of facial herpes following the relief of the attack she had had in February; further attacks had followed, however.

On November 10, 1936, further examinations by cystoscopy, pyeloscopy and pyelography showed that indigo-carmine injected intravenously was secreted in eight minutes from the left kidney and in eleven minutes from the right kidney. The left renal pelvis, when injected with sodium iodide (15%), had held fifty cubic centimetres with pain. Dr. Fitzpatrick said that after ten minutes with the ureteric catheter withdrawn no diminution of the left shadow had occurred, and this indicated an abnormally slow emptying time. Eserine, one one-hundredth of a grain, was injected intramuscularly, and active contraction of the calyces and pelvis was seen and the density of the pelvic shadow lessened rapidly.

Dr. Fitzpatrick suggested that the case was probably one of those which S. H. Harris had termed "renal sympathetico-tonus" or the "painful motility syndrome". Having proved the absence of an organic ureteral stricture and negative cultural and microscopic findings in the urine, Harris had explained the hydronephrosis as due to over-action of the sympathetic nerves causing spasm of the ring muscles, which had been shown by Kelly and Bowman to exist at the outlets of the calyces and renal pelvis. Dr. Fitzpatrick said that this was similar in conception to the hypothesis of sympathetico-parasympathetic imbalance in cardiospasm and Hirschsprung's disease *et cetera*. At the same time there was some inhibition of peristalsis proximal to the sphincter apparatus. Harris considered that overwork, worry and a hyperexcitable sympathetic nervous system were the predisposing factors; and all the factors had been present in the patient shown by Dr. Fitzpatrick. The patient was definitely an allergic subject, and her initial attacks of renal colic had begun during the month of November, when she had started to show sensitiveness. Dr. Fitzpatrick wondered if this could indicate a possible relationship in ætiology between her painful renal symptoms and her allergy; in other words, was her sympathetico-parasympathetic imbalance one of the underlying causes of her allergic reactions?

DR. A. E. COATES considered that the evidence brought forward by Dr. Fitzpatrick supported a diagnosis of hydro-

nephrosis of neurogenic origin. There was, however, an absence of ptosis of the left kidney, and the patient was not of the usual sympatheticotonic type. The skin lesion appeared to Dr. Coates to be a long-standing condition not related to autonomic imbalance. The patient had had eight children and was passing through the early stages of the menopause. The relation of the attack of renal pain to the menses might be significant. While due allowance should be made for all the evidence of sympathetic imbalance, Dr. Coates could not help thinking that there might be some factor of pelvic origin responsible for the attacks. He suggested that before an operation on the renal sympathetic nerves was undertaken it would be wise to exclude carefully gynæcological disorders which might be responsible for the condition. In any case the operation was not without some risk; the physiological basis for it was still *sub judice*, and the patient was relieved by the injection of eserine. She was not disabled, except during her attack of pain. A policy of conservatism for the time and perhaps some research into the possible pelvic anatomical or physiological abnormalities related to the condition would repay the investigator and add to knowledge. If no benefit accrued from this line of inquiry in treatment, then sympathectomy would be a justifiable procedure.

DR. H. I. HOLMES said that as the patient was tender in the left side of the pelvis and had had attacks of renal colic, he wondered whether some ureteric stenosis was going on. He too considered that gynæcological causes should be excluded before anything else was undertaken.

DR. G. A. PENINGTON suggested that an intravenous pyelogram would help to decide the site of any obstruction present.

DR. K. ROSS said that he thought that Dr. Fitzpatrick had proved his case according to the conditions which had been set out by Harris. Dr. Ross pleaded guilty to having removed some of these kidneys. In one such case a similiar condition had developed in the other kidney.

Dr. Fitzpatrick, in reply, said that he had tried to exclude ureteric stricture in this case. He had had three or four patients, the subjects of hydronephrosis, without calculus or stricture, and until he heard Harris's explanation he had been unable to give any reason for the condition. The patient he had shown had suffered intense pain in the attacks and was liable to call him out in the middle of the night. Even half a grain of morphine would not relieve the pain completely. The patient would not agree to go on suffering such pain if she could be freed from it. The subsequent course of the case would be full of interest for him. He would go into the gynæcological history thoroughly and make sure of the ureter before he undertook any operation on the sympathetic nerves.

Congenital Dislocation of the Hip.

DR. KENNETH F. O'DONNELL showed a girl, seventeen years of age, with congenital dislocation of the left hip and formation of a secondary socket. The patient had complained of a pain in the left hip two months before the meeting, and of an aching back, with an increase of the limping. She was the second child of the family; a breech presentation, there had been difficulty with the legs and the child had been late in walking. She had started to walk at three years and the mother had noticed a depression in the left hip region, but had not thought anything of it; the limp continued, but the child had been active and well, and medical advice was not sought until 1932, when a skiagram revealed a dislocation with a secondary socket; no treatment had been carried out. The patient had remained without pain and led an active life until two months before the meeting, when she again sought medical advice. She had had no other illness, excepting an attack of hæmaturia one year before, and no investigation was carried out at the time.

On examination the characteristic gait was evident, with scoliosis and lordosis of the lumbar spine and a shortening of three centimetres. A skiagram revealed the change in the head of the femur, the narrow acetabulum and the newly formed socket, with changes in the ilium. Dr. O'Donnell invited discussion as to whether any surgical aid was possible or desirable, and concerning the type of boot advisable to overcome the deformity and to prevent any further pelvic distortion. With the boot raised one and a quarter inches she was getting more pain than she should, and he invited discussion as to whether compensation should be complete.

DR. DOUGLAS GALBRAITH said that he was better acquainted with this condition in younger children. The patient seemed to have formed a good acetabulum and there was very little, if any, secondary arthritis. With reference to the late diagnosis of this condition, Dr. Galbraith recalled the case of a girl of about eight years of age, shown at a meeting of the Melbourne Pædiatric Society by the late Dr. Mervyn Stewart; bilateral congenital dislocation of the hips was discovered at an examination which was carried out to determine why the child could not "do the splits" properly. There were many points about congenital dislocation of the hip which caused concern to the members of the surgical staff at Frankston; one of them was the extraordinary difficulty of getting good results when the children came under treatment at the later ages. Dr. Galbraith also referred to the case of a woman of over sixty years of age, attending at the Alfred Hospital, who had congenital dislocation of the hips which had caused her no inconvenience.

DR. H. BOYD GRAHAM said that he hesitated to express a surgical opinion, but he knew that manipulative interference with congenital dislocation of the hip was fraught with great difficulty unless it was first undertaken in infancy; he thought that his surgical colleagues considered these patients rather old for reduction, say, at three years of age. In the patient shown by Dr. O'Donnell deep lordosis was present and the pain seemed to be chiefly lumbar. She took her weight on the ball of the foot instead of dividing it between the ball and the heel. Dr. Graham suggested that close attention should be paid to postural exercises, and with improvement of muscle tone and body mechanics, which could be brought about only by equalizing the length of the two legs by means of a special boot, it might be possible to overcome her deformity without interfering with the hip joint. Young women objected to these boots as unsightly, but if the advantages to her gait and for the avoidance of future disabilities were pointed out to her carefully, it was likely that she would overcome her prejudice to the wearing of the boot. Dr. Graham also mentioned the advisability of raising the boot gradually instead of making a full correction in the first instance.

DR. F. KINGSLEY NORRIS said that it would be a good thing to make a routine radiological investigation of the pelvis and hip joints of every baby, and that the aim of treatment in congenital hip disease was to restore a movable hip in the site of the normal hip joint. At times complete restoration was disappointing; one could not tell whether the head of the bone would go in or not. Only that week he had seen at an open operation a distorted, contracted capsule present, filling the acetabulum and offering the obvious explanation for the want of success of the previous manipulations. Dr. O'Donnell's patient had postural spinal pain. The bone was resting against a very good shelf in a very good false acetabulum, and she had very good movement; he would advise Dr. O'Donnell to carry on with the boot.

DR. S. C. FITZPATRICK commented on the obstetrical aspect of the case, but thought that with unilateral dislocation only there was no need to worry very much about future obstetrical difficulty. He thought that the onset of pain in the case of the patient shown by Dr. O'Donnell was an evidence of the objection of the patient to the raising of the boot on the short side, and that it would disappear on adjustment of the boot.

Dr. O'Donnell, in reply, thanked those who had contributed to the discussion and stated that he thought the patient was getting some true pain in her hip, which was causing him anxiety in case it was due to early arthritis; he thought that her obstetric future was satisfactory.

Hydatid Disease.

DR. J. LE M. KNEEBONE read a paper entitled "Hydatid Disease: A Survey of Sixty Cases" (see page 201).

DR. H. BOYD GRAHAM read a paper entitled "Hydatid Disease in Children" (see page 206).

DR. KEITH D. FAIRLEY congratulated both speakers on their most interesting papers. Since treatment was surgical, diagnosis had, of course, been of most interest to the physician. Dr. Fairley said that the only pathognomonic clinical sign of hydatid disease was the hydatid thrill, which was seldom encountered. He said that uncomplicated and degenerated cysts produced symptoms purely through the mechanical effects of pressure. In the majority of cases this disease was latent throughout life. Dr. Fairley said that in two series of several thousand autopsies performed by Professor Cleland in Adelaide and Dr. Wright Smith in Melbourne, the incidence of latent hydatid disease, chiefly in the form of degenerated cysts in the livers of elderly patients, was from 2·5% to 3·0%. Cysts in the liver often reached a huge size before producing symptoms. In the brain, however, pressure symptoms appeared early, hence the greater incidence of cysts in this site in childhood. Dr. Fairley said that cysts in the lung were prone to the complications of rupture and suppuration at a relatively early stage, and this was the reason, as Dr. Graham had suggested, for the greater apparent incidence of pulmonary cysts in children. Repeated attacks of "pneumonia" and allied pulmonary complications should arouse suspicion of some underlying cause, such as a pulmonary cyst. Cysts in the lung were most frequently mistaken for tuberculosis, and in the liver for gall-stones. The immediate Casoni reaction was of great value in diagnosis, but care was needed in its interpretation and in the technique of the test. Dr. Fairley said that positive results were obtained in about 75% of cases in hydatid disease; they were also found in some cases with other helminthic infestations, past or present, as, for instance, in a patient he had seen a few days before the meeting, who had suffered from hookworm disease for some years previously. Failure to react was very valuable in excluding hydatid disease in approximately 97% of cases. This test was useless in the diagnosis of any recurrent or residual cyst, since, once this reaction appeared, either before or after a first operation for this disease, it might persist for several decades in the absence of any clinical evidence of further infestation. The complement fixation test gave positive results in about half the cases of hydatid disease. It was more specific than the Casoni reaction, at least in regard to past infestation with other helminths.

Dr. Fairley said that its great value was in the diagnosis of recurrent or residual cysts. The fixation of six or more minimal hæmolytic doses of complement nine or more months after operation indicated the presence of further cysts. He said that the failure to react was of no value in excluding hydatid disease. The detection of a raised eosinophile count occasionally pointed the way to a correct diagnosis. When the complement fixation test was not available, the simple precipitin test, carried out with carbolized hydatid fluid, was very valuable. Dr. Fairley said that, depending on the site of the cyst, hydatid disease might simulate almost as many diseases as syphilis. Once the clinician considered the possibility of hydatid disease, the examination of discharges for hydatid material and the use of immunological tests and skiagraphic investigation enabled the presence or absence of hydatid disease to be accurately determined in about 90% of the cases. The greater incidence of hydatid cysts in the right as compared with the left lobe of the liver, which Dr. Kneebone had mentioned, was probably related to the greater size of the right lobe, and possibly also to the two streams of blood in the portal vein. McNee had stated that the blood from the spleen went mainly to the left lobe of the liver, while that from the gut passed to the right lobe.

DR. S. C. FITZPATRICK said that as the hour was late he would make reference to two points only: the surgical treatment of liver cysts and the significance of distortion of cysts, particularly in the lungs. In the more usual transpleural operation on cysts of the right dome of the liver with infection the sutures were liable to cut and run, and vomiting led to strain on the sutures. The rule was to open as far forward and as low down as possible, but the right dome took a deep dip at the back. H. Constantini used an incision along the right costal margin into the peritoneal cavity and one along the intercostal space nearest the cyst; he placed his left hand up beneath the diaphragm and pressed it outwards; then he sewed the parietes, pleuræ and diaphragm together and opened down to the cyst; this provided a steadier field for sewing, but did not do away with the strain on the needle holes afterwards. Dr. Fitzpatrick suggested that the subpleural route was more satisfactory. By œdematizing or ballooning the tissues with infiltration anæsthesia it was possible to raise slowly and deliberately the parietal pleura, the costo-phrenic reflexion and then the diaphragmatic pleura successively, uncover the radiating muscle fibres of the diaphragm and split them to reach the peritoneum on the dome of the liver. Dr. Fitzpatrick had first used this method successfully to drain a suppurating right liver dome cyst in a male of seventy-two years of age without any subsequent pleural infection. Dr. Fitzpatrick had observed that in skiagrams of the lungs a distorted appearance of hydatid cysts was frequently to be seen; by this he meant that the shape of the cyst might be other than spherical. Operative findings on such cysts had led Dr. Fitzpatrick to conclude that the distortion of the cyst gave a clue to its geography. He suggested that the ovoid, elongated appearance of a cyst indicated that part of its surface had reached the interlobar fissure; that the emerged cyst would show flattening on its parietal aspect; that the secondary pleural cyst would show elongation parallel to the ribs immediately external to it; and that the intramuscular cyst would show elongation parallel to the muscle fasciculi. Dr. Fitzpatrick added that it was hardly necessary to say that the exact geography of a lung cyst and the relation to the pleura were of great assistance to the surgeon when he was deciding the method of approach and the surgical treatment which would give the best results. He had also noted that centrally placed cysts would come to the surface sooner or later; so an uncomplicated cyst which was inaccessible without doing great damage to the lung was worth watching, and operation might well be delayed until the cyst had emerged to a position in which it could be approached more safely. He had taken a series of skiagrams of such a cyst and would be pleased to show them to anyone interested.

DR. KENNETH O'DONNELL referred in particular to some of the features of interest to the anæsthetist at operations on hydatid cysts. In the western district one was always on the look-out for the first symptoms of severe complications of the cyst. It was usual to use local anæsthesia at times, combined with nitrous oxide and oxygen. For the operations on pulmonary cysts positive pressure apparatus was of great value. The grave and worrying collapse that so frequently occurred after the evacuation of the cyst gave the anæsthetist great concern, but the anaphylactic phenomena should be eliminated under general anæsthesia.

NOMINATIONS AND ELECTIONS.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Kellett, William Hessel, M.B., B.S., 1935 (Univ. Sydney), 39, East Esplanade, Manly.
Skype, Joseph Michael, M.B., B.S., 1935 (Univ. Sydney), Newcastle Hospital, Newcastle.

Obituary.

RAYMOND BALLS.

WE regret to announce the death of Dr. Raymond Balls, which occurred on January 27, 1937, at Mosman, New South Wales.

Honours.

NEW YEAR HONOURS.

HIS MAJESTY THE KING has been pleased to confer the following honours on medical practitioners:

Dr. George Moncrieff Barron, of Sydney, has been created an Officer of the Most Excellent Order of the British Empire.

Dr. Sydney Letts Dawkins, of Adelaide, has been created an Officer of the Most Excellent Order of the British Empire.

The congratulations of the medical profession are extended to Dr. Barron and Dr. Dawkins.

Congress Notes.

HOTEL ACCOMMODATION.

THE Executive Committee of the fifth session of the Australasian Medical Congress (British Medical Association), to be held at Adelaide from August 23 to 28, 1937, has forwarded the information regarding hotel accommodation that is set out in the table below.

This information is believed to be correct, but the Accommodation Committee does not hold itself responsible for any inaccuracy, or for the rates or tariffs shown.

Intending visitors are recommended to make reservations direct with the hotel managers beforehand, stating fully the type of accommodation required and the dates. This may also be arranged through the Secretary of the Accommodation Committee (Dr. M. T. Cockburn, 170, North Terrace, Adelaide).

The "Accommodation for" column shows the maximum normal capacity of the premises, but naturally the hotels are prepared to make only a proportion of their accommodation available during congress week.

Proceedings of the Australian Medical Boards.

SOUTH AUSTRALIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Acts* 1919 *to* 1921, of South Australia, as duly qualified medical practitioners:

Welch, F. E., M.B., B.S., 1936 (Univ. Adelaide), Adelaide.
White, J. H., M.B., B.S., 1935 (Univ. Adelaide), Adelaide.
Ray, J., M.B., B.S., 1936 (Univ. Adelaide), Adelaide.
Chapple, C. F., M.B., B.S., 1936 (Univ. Adelaide), Adelaide.
Heard, K. H., M.B., B.S., 1936 (Univ. Adelaide), Adelaide.
Frayne, J. F., M.B., B.S., 1936 (Univ. Adelaide), Adelaide.
Kirtle, Patricia, M.B., 1931 (Univ. Sydney), Northfield.

Books Received.

A PRACTICAL MEDICAL DICTIONARY, by T. L. Stedman, A.M., M.D.; Thirteenth, Revised Edition; 1936. London: Baillière, Tindall and Cox. Royal 8vo, pp. 1303, with illustrations.

AN INTRODUCTION TO GENERAL PRACTICE, by E. Kaye Le Fleming, M.A., M.D.; 1936. London: Edward Arnold and Company. Crown 8vo, pp. 150. Price: 5s. net.

	Name of Hotel.	Address.	Accommodation for.	Tariff from Bed and Breakfast.	Tariff from Per Day.	Tariff from Per Week.
				s. d.	s. d.	£ s. d.
ABCDEF	South Australian ..	North Terrace, Adelaide	120	12 6	21 0	7 0 0
ABCDE	Hotel Richmond ..	128, Rundle Street .. .. .. (Bedroom with private bathroom)	80	12 0 13 6 17 6	17 6 20 0 25 0	
ABCDEF	Hotel Napoleon ..	King William Street	35	10 6	14 6	4 10 0
BCDE	Hotel Botanic	North Terrace	30	10 0	16 0	4 10 0
ABCDE	Hotel Ambassadors ..	King William Street	42	10 6 12 6	17 6 20 0	4 4 0 4 10 0
ABCDE	Oriental	Rundle Street	50	10 6 12 6	17 6 21 0	5 12 6 6 10 0
BE	Majestic	King William Street	30	8 6	10 6	3 3 0
BE	Southern Cross	King William Street	35	8 0	10 6	3 3 0
ABE	Hotel Rundle	Rundle Street	30	—	12 0	4 4 0
ABE	Hotel Adelaide	Pirie Street	35	7 0	10 0	3 3 0
E	Black Bull	Hindley Street	35	6 0	10 0	3 3 0
ABCDEF	Grosvenor (unlicensed)	North Terrace	310	6 0	10 0	3 2 6
E	Victoria	94, Hindley Street	42	5 6	8 6	2 10 0
		Northern Suburb.				
E	Cathedral	45, Kermode Street, North Adelaide..	20	6 0	10 0	3 3 0
		Seaside Resorts.				
E	Pier Hotel	2, Jetty Road, Glenelg	90	10 6 10 6	12 0 14 0	3 10 0 4 4 0
BCE	Hotel St. Vincent ..	28, Jetty Road, Glenelg	40	7 6 8 6	12 0 15 0	4 4 0 5 5 0
	Largs Pier	Esplanade, Largs Bay..	28	6 0	10 6	3 3 0

A=Bedrooms with private bathrooms.
B=Hot and cold running water in bedrooms.
C=Bedrooms with private telephones.
D=Reading lamps in bedrooms.
E=Hot and cold running water in bathrooms.
F=Rooms with private bathrooms extra.

HISTORICAL NOTES ON PSYCHIATRY, by J. R. Whitwell, M.B.; 1936. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 264. Price: 10s. 6d. net.

THE DIGESTIVE TRACT: A RADIOLOGICAL STUDY OF ITS ANATOMY, PHYSIOLOGY AND PATHOLOGY, by A. E. Barclay, M.A., M.D., D.M.R. and E., M.R.C.P., F.A.C.R.; Second Edition; 1936. London: Cambridge University Press; Melbourne: S. Jaboor. Crown 4to, pp. 462, with illustrations. Price in England: 36s. net.

Diary for the Month.

FEB. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 16.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 24.—Victorian Branch, B.M.A.: Council.
FEB. 25.—South Australian Branch, B.M.A.: Branch.

Medical Appointments.

Dr. J. A. Rolland has been appointed a Member of the Opticians Board of South Australia.

* * *

Dr. J. W. Rollison has been appointed Medical Superintendent of the Adelaide Hospital.

* * *

Dr. H. A. McCoy has been appointed Honorary Assistant Radiologist at the Adelaide Hospital, Adelaide.

* * *

Dr. D'A. N. Short has been appointed Government Medical Officer at Millthorpe, New South Wales.

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Dr. E. S. Morris has been reappointed President of the Board of Health of New South Wales.

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Dr. G. W. Howard has been appointed Acting Deputy Superintendent of the Northfield Mental Hospital.

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Dr. J. W. B. Bean has been appointed Medical Officer, Department of Public Instruction, Brisbane.

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Dr. D. M. Seeley has been appointed, pursuant to the provisions of the *Workers' Compensation Act*, 1928, of Victoria, Certifying Medical Practitioner and Medical Referee at Mildura, Victoria.

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The undermentioned appointments have been made in the Inspector-General of Hospitals Department, Adelaide: Dr. W. L. Jeffries, Director-General of Medical Services, Inspector-General of Hospitals, under the provisions of the *Mental Defectives Act*, 1935, and Chairman of the Nurses' Board of South Australia, under the provisions of the *Nurses Registration Act*, 1920; Dr. J. G. Sleeman, Chief Medical Officer for Tuberculosis Services; Dr. H. McI. Birch, Superintendent of Mental Hospitals; Dr. R. S. Rogers, Deputy Superintendent, Enfield Receiving House; Dr. G. H. Howard, Visiting Medical Officer, Enfield Receiving House.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xx, xxi, xxii.

DEPARTMENT OF PUBLIC HEALTH, WESTERN AUSTRALIA: Junior Resident Medical Officer.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Officers.

THE RACHEL FORSTER HOSPITAL FOR WOMEN AND CHILDREN, SYDNEY, NEW SOUTH WALES: Resident Medical Officer, Part-Time Medical Registrar, Honorary Assistant Ophthalmic Surgeon.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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